THE
BRITISH JOURNAL OF SURGERY

VOL. XVII. OCTOBER, 1929. No. 66.

EPOCH-MAKING BOOKS IN BRITISH SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

X. HUNTER'S OPERATION FOR THE CURE OF ANEURYSM.

So obsessed have modern English surgeons become by the name of Lister that it would be difficult for many of them to say off-hand why Hunter is looked upon, by those who know, as the greatest of surgeons. He was a bad lecturer, a confused thinker, and a very indifferent writer, yet he found surgery mediæval and clinical; he left it a science. How was it done? Go to the museum he collected and let each one see for himself. Study it and the whole science of surgery is displayed in terms of morbid anatomy, comparative as well as human. Many surgeons before him had collected pathological specimens and made museums, but they had used them solely for teaching purposes and they had passed by sale from teacher to teacher. Hunter alone collected not to teach but to learn, and comparing like with like he thought about them and drew wholly fresh conclusions. A few of his contemporaries and the most receptive of his pupils entered into his spirit. To them he was 'the Dear Man'; they had penetrated his shyness and quarrelsomeness, and to them he was a personality ever urging them to follow out his lines of thought. He thus became the founder of a great school of scientific surgery which owed as much to his successors as to himself for its widepsread influence both at home and abroad.

Hunter's work was encyclopædic, for his curiosity was insatiable. We are concerned only with the surgical side, and it is difficult to make a selection. The treatise on inflammation, divested of its rugged style, contains much food for thought and foreshadows many of the surgical discoveries afterwards made possible by advances in chemistry and in physics. The essay on gunshot wounds, based upon only a short experience of actual war, taught that such wounds are not things apart, but should be treated on the same principles as govern other kinds of wounds. The pamphlet upon venereal diseases proved of value, although it was based upon incorrect

premisses and the conclusions were wrong; yet it served its purpose because it led to a very thorough examination of the diseases by those who wished to refute the author.

It is interesting to follow up the line of procedure which Hunter adopted when he desired to unravel a new idea, and this can be done with tolerable ease in the case of his operation for the cure of popliteal aneurysm.

On May 22, 1788, Mr. Edward Ford, living in Golden Square and Surgeon to the Westminster Dispensary, which afterwards became the Westminster Hospital, communicated to the *London Medical Journal* a letter "On Cases of the Spontaneous Cure of Aneurism". The first history was that of John Cathay, age 36, of Great St. Andrew's Street, Seven Dials, who had an aneurysm of the left popliteal artery which disappeared, though he died afterwards of a right femoral aneurysm. Hunter had seen the case during life as early as September, 1785. He attended the post-mortem examination, secured the specimen, and had it carefully drawn. The second history is that of a man in whom a large popliteal aneurysm was so far cured by prolonged rest that he could walk ten miles without harm. This case Hunter had also seen. Ford was opposed to operation, whether by amputating the limb or tying the artery, on the ground that it usually ended in the death of the patient. He pointed out that the cure by nature is permanent; that the inert mass left behind is not likely to produce any mischief; that the unsuccessful event of the operation for the popliteal aneurysm does not depend principally on any particular hazard in consequence of an obstructed circulation in the ham, but upon other causes; and, most important of all, that if the communicating branches above the tumour are large enough to carry on the circulation in the extremity, the patient may recover, but if they are not, a mortification must of course ensue.

These conclusions were probably formulated after consultation with Hunter, who was living near by in Leicester Square. At any rate, after talking the matter over with Ford and carefully examining the piece of artery which had become converted into what would now be called fibrous tissue, he set to work experimentally. The story is continued by Everard Home in 1793 shortly before the death of his brother-in-law. He says: "Mr. Hunter finding an alteration of structure in the coats of the artery previous to its dilatation and that the artery immediately above the sac seldom unites when tied up in the operation for the aneurism, so that as soon as the ligature comes away, the secondary bleeding destroys the patient, was led to conclude that a previous disease took place in the coats of the artery in consequence of which it admitted of dilatation capable of producing aneurism. But not satisfied with the experiments on frogs, given by Haller in support of the opinion that weakness alone was sufficient to produce the dilatation, he resolved to try the result in a quadruped, which, from the vessels being very similar in their structure to those of the human subject, would be more likely to ascertain the truth or fallacy of Haller's opinion.

"Mr. Hunter laid bare the carotid artery of a dog for above an inch in length and having removed its external coat and afterwards dissected off the other coats layer by layer till what remained was so thin that the blood was plainly to be seen through it, left the dog to himself. In about three weeks

the dog was killed and the parts examined, when it appeared that the two sides of the wound having closed upon the artery, the whole of the surrounding parts were consolidated, forming a strong band of union, and the artery itself was neither increased nor diminished in size.

"This experiment appeared very conclusive, as the coats of the artery were weakened to a much greater degree without dilatation than can ever happen from accident in the living body, independent of morbid affection. But it was objected on the other hand, that the parts having been left to themselves, immediately closed upon the weakened portion of the artery, and, being cemented together by the coagulated blood, effectually secured it against any dilatation. To try the force of this objection, I [i.e., Everard Home] made the following experiment.

"I laid bare the femoral artery of a dog, about two inches below Poupart's ligament, for about an inch in length and dissected off the coats till the hæmorrhage from the vasa vasorum was considerable, and the circulating blood was distinctly seen through the internal membrane of the artery. The hæmorrhage soon stopped by exposure, the surface was wiped dry and afterwards covered with a dossil of lint to prevent the sides of the wound from uniting. The dog continued very well, and the wound healed up from the bottom; after six weeks the dog was killed and the artery was injected, that it might be examined with greater accuracy. It was not perceptibly enlarged or diminished and its coats at this part had recovered their natural thickness and appearance.

"The results of these experiments confirmed Mr. Hunter in his opinion that the artery, in cases of aneurism, is in a diseased state and led him to believe that the disease often extends along the artery for some way from the sac; and that the cause of failure in the common operation arises from tying a diseased artery, which is incapable of union in the time necessary for the separating of the ligature. The femoral and popliteal arteries are portions of the same trunk, presenting themselves on different sides of the thigh, and are readily come at in either situation; but where the artery is passing from the one side to the other, it is more buried in the surrounding parts and cannot be exposed without some difficulty.

"In performing the operation for the popliteal aneurism, especially when the tumour is large, the ligature is commonly applied on the artery at that part where it emerges from the muscles. This mode of performing the operation will be found inadequate if the disease of the artery extends above the sac; for if the artery should afterwards give way, there will not be a sufficient length of vessel remaining to allow of its being again secured in the ham. To follow the artery up through the insertion of the triceps muscle, to get a portion of it where it is sound, becomes a very disagreeable part of the operation; and to make an incision upon the fore part of the thigh, to get at and secure the femoral artery would be breaking new ground; a thing to be avoided, if possible, in all operations.

"Mr. Hunter, from having made these observations, was led to propose, that in this operation the artery should be taken up in the anterior part of the thigh, at some distance from the diseased part, so as to diminish the risk of hæmorrhage and admit of the artery being more readily secured should

any such accident happen. The force of the circulation being thus taken off the aneurismal sac, the progress of the disease would be stopped; and he thought it probable, that if the parts were left to themselves, the sac with its contents, might be absorbed and the whole of the tumour removed which would render any opening into the sac unnecessary.

"Upon this principle Mr. Hunter performed the operation at St. George's Hospital."

The first operation was performed upon a coachman, age 45, in December, 1785, and the patient died on April 1, 1787, fifteen months after the operation. A post-mortem examination was obtained with difficulty and it was found that the aneurysmal sac had shrunk and was filled with laminated clot.

Hunter performed the operation five times and with sufficient success to show the correctness of his theory that: (1) Slowing of the blood-stream would cure an aneurysm; (2) Aneurysm was caused by disease of the artery and was not the simple result of long-continued local injury; (3) So long as there was sufficient collateral circulation gangrene would not result from ligature of the main artery—amputation therefore was unnecessary.

DERMOID CYST OF THE MEDIASTINUM.

By Sir CARRICK ROBERTSON,
HONORARY SURGEON TO THE AUCKLAND HOSPITAL, NEW ZEALAND.
AND R. E. BEVAN BROWN,
ASSISTANT DIRECTOR IN THE PATHOLOGY DEPARTMENT, AUCKLAND HOSPITAL.

On consulting the literature we find records of 123 cases of mediastinal dermoids. This number is small when we consider that the condition is an arresting one and on this account more likely to be reported, and that this number comprises all the cases recorded over many years in Europe and America. Consequently we feel that the occurrence of such a case is worthy of mention.

CASE HISTORY.

The patient, Mrs. W., age 36, was admitted to the Auckland Hospital on June 11, 1928. She was described as having suffered as a child from a 'weak chest'; at the age of 14 she contracted rheumatic fever, followed by chorea, which lasted

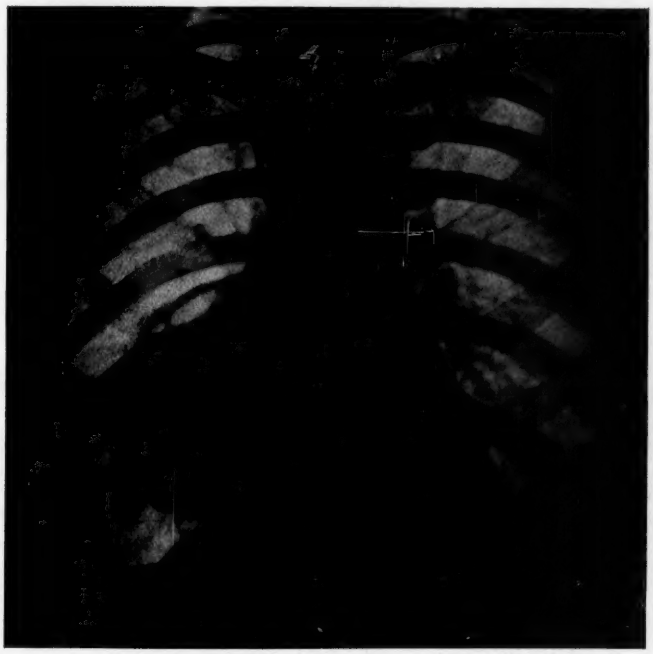


Fig. 129.—Antero-posterior radiogram of chest before operation.

several years, and she was more or less an invalid up to the age of 22. For the last ten years her menstrual history had been normal and healthy, but prior to that periods were scanty and irregular. Patient was a nullipara.

Three years prior to admission she began to have severe paroxysms of coughing, which came on every few weeks. No expectoration followed till eighteen months before admission, when she coughed up a hair. Since then she occasionally coughed up a few hairs during a paroxysm. Three months before admission after a severe bout of coughing she brought up a number of coarse white hairs about three inches long. After this on various occasions she coughed up hairs and sebaceous matter. A fortnight before admission she consulted Dr. Horton, St. Heliers, to whom we are indebted for much clinical information: she had a very severe attack of coughing, with pain in the back and below the right breast. The day before her temperature was 101°, and she was coughing up yellow granules and several hairs. There were no abnormal physical signs in the chest. Temperature fell to normal and the pain was relieved, but she continued to cough up sebaceous material.

FIG. 130.—Lateral radiogram of chest before operation.

Dr. F. J. Gwynne, radiologist, was consulted, and reported as follows (*Figs.* 129, 130): "In the anterior mediastinum, on the right of the midline, there is a rounded, well-defined, abnormal shadow. It extends between the second and fourth rib-cartilages and is about three inches in diameter. In the erect position the upper quarter containing air is separated from the lower part of the obscurity by a definite fluid level. The findings are consistent with the diagnosis of dermoid cyst of the mediastinum."

At the same time a sample of the sputum was submitted to one of us (R. E. B. B.). The specimen consisted of yellow sebaceous material and a few hairs. Microscopically it showed a few epithelial cells, and enormous numbers of bacteria. No pus cells were seen. Cultures grew *B. coli communis* and diphtheroid bacilli, suggesting a heavy infection but one of relatively low virulence. It was reported that all the evidence pointed to the presence of a dermoid cyst of the mediastinum, which had become infected.

Radiological, laboratory, and clinical findings left no room for doubt. The finding of hairs is regarded as conclusive (though

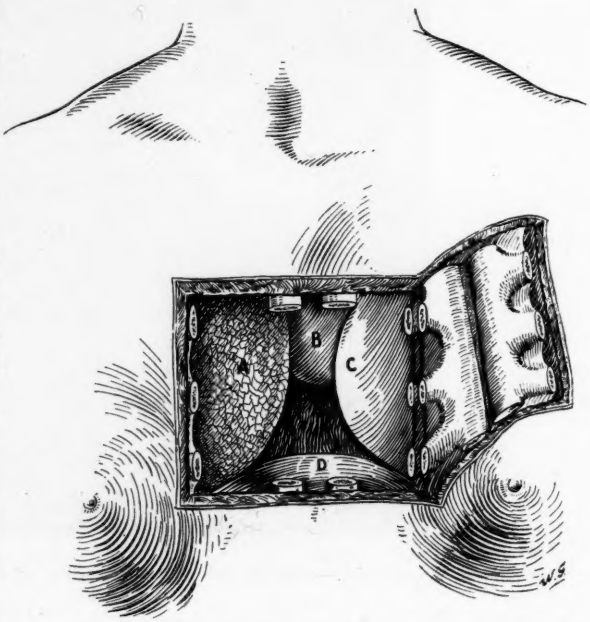


FIG. 131.—Semi-diagrammatic view of the tumour and its relations. Note the bifid sternum. A, Right lung; B, Dermoid; C, Pericardium; D, Diaphragm.

apparently this is a rare occurrence, only eight previous cases of the kind being recorded). No other condition than that of a dermoid cyst rupturing into a bronchus could account for the facts. Radical surgical treatment was regarded as imperative, as, if left alone, the cyst might exert grave pressure symptoms on vital structures, and if through rupture its highly infected contents escaped into the mediastinum, fatal suppuration was almost certain to be the result. Consequently operative measures were decided upon, and carried out by one of us (C. R.) in two stages.

At the first stage (June 15), a trap-door consisting of the second, third, and fourth costal cartilages of the right side and the corresponding piece of the sternum was lifted up. The flap was made to hinge on the left side by dividing the left costal cartilages with a knife. Both the right and left internal mammary arteries were secured and tied. A hard-walled cyst was now felt, deeply placed in the mediastinum. The appearance and relations of the cyst can be seen in *Fig.* 131.

Unfortunately manipulation of the trap-door at this stage resulted in a perforation of the pleura on both sides. The patient immediately collapsed, as no

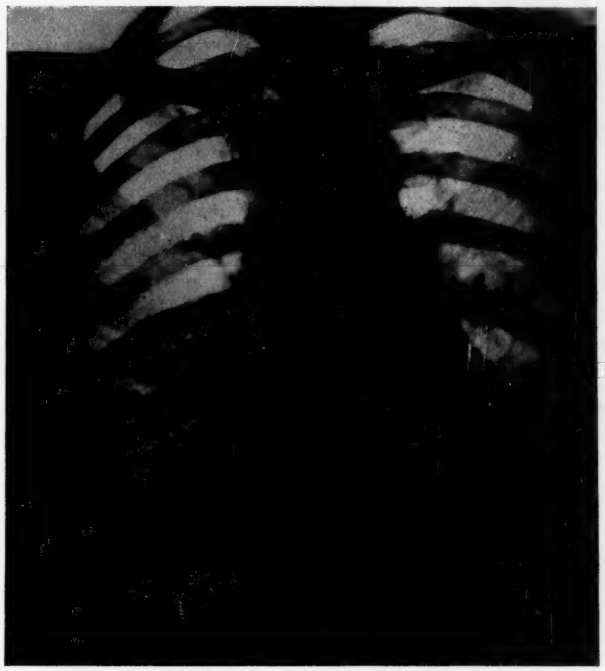


FIG. 132.—Antero-posterior radiogram of chest after operation.

air entered the trachea. Dr. Gould, the anæsthetist, promptly passed an intratracheal catheter and pumped in oxygen. In the meantime the flap was hurriedly sealed down to prevent further air from entering the pleural cavities. Gradual improvement took place, but largely owing to the unstable and excitable mental condition of the patient, convalescence was not a tranquil process for some days.

The second stage was performed on July 6. On this occasion Dr. Gould administered intratracheal ether from the beginning. The trap-door was lifted up, and the cyst was then seen as a whitish round mass, partly covered by and firmly adherent to the anterior border of the right lung. On the left side the pericardium over the right auricle was closely adherent, and covered this part of the cyst. The left innominate vein was adherent to the upper part of the cyst. A patient dissection was now carried out, and as there was no line of cleavage between the cyst and surrounding structures, progress was slow. Considerable difficulty was encountered :

the cyst wall was friable and broke away in many places; posteriorly the cyst extended well into the posterior mediastinum. Eventually it was all removed. No sign of the bronchial fistula was apparent. The cyst was about the size of a small coconut. A drainage tube was put into the depths of the wound and a suction apparatus attached thereto. A free discharge of blood-stained fluid continued for some hours, but the suction drainage removed it before it caused any untoward pressure symptoms. It is almost certain that the pericardium was opened in the region of the right auricle and that the right pleura was again perforated, but the suction apparatus, acting continuously, prevented the serous exudate from the large raw area flowing into these cavities. Our feeling was that the suction drainage was a great factor in the patient's recovery. Save for some superficial sepsis, which for some weeks delayed the healing of the wound, recovery was uneventful. The patient's temperature was normal from the sixth day. She was discharged on Aug. 16. On Oct. 9 she appeared before the Auckland Clinical Society. She was in excellent health, all the symptoms had disappeared, and she complained of nothing at all save occasional tachycardia. An X-ray photograph taken at this time (*Fig.* 132) shows a practically normal chest.

DISCUSSION.

There is a considerable literature relating to dermoid cysts of the mediastinum.

Age Incidence.—If we take the age at the time of operation or death as the age of incidence, cases commonly occur between the ages of 15 and 35, i.e., mainly in young adult life. Usually, however, there have been vague symptoms pointing to a much longer history—to puberty or much earlier. As there is an embryological explanation of their occurrence one might expect symptoms from birth, but of course the cyst may not start to grow till much later—e.g., at puberty. For all that, dermoid cysts and teratomata of the mediastinum have been noted in infants. The history of 'weak chest' during childhood in the case of our patient points to a similarly early origin.

Symptoms and Signs.—There is general agreement that symptoms and signs may be variable or slight. The onset may be insidious, and in a number of recorded cases no tumour was suspected before autopsy. Where symptoms and signs are present they are usually related to the respiratory system; cough, dyspnœa, and free expectoration are relatively common. Hæmoptysis occurs in a few cases. The coughing up of sebaceous material, through rupture into a bronchus, is sometimes seen: more rarely hairs are coughed up at the same time, which is diagnostic. Christian mentions one case of dysphagia.

Pain (felt locally) is relatively rare, and venous engorgement of the neck due to intrathoracic pressure still more so. In one or two cases a visible tumour has appeared above the sternum. Febrile symptoms and general malaise may of course occur. Commonly the diagnosis is made of some pulmonary or pleural disease—e.g., encysted empyema, pulmonary tuberculosis, bronchiectasis, or tuberculosis of mediastinal glands; sometimes the tumour is thought to be an aneurysm of the aortic arch or an hydatid cyst of the lung. It is said that X-ray appearances are characteristic, and such is our experience in this case.

Course.—The general opinion seems to be that most patients succumb in one to four years from the onset of symptoms unless surgical intervention takes place. We have already referred to the dangers of neglecting the

condition; the cyst may increase in size, and the effects of its pressure on the heart, great vessels, lungs, trachea, or other structures may be serious. If rupture into a bronchus takes place, infection is almost bound to ensue, and such infection involves the grave risk of a mediastinitis. Some writers declare the cyst may become malignant. We do not know how to assess this danger; it may be that such malignant tumours were malignant in the first instance, but we cannot dismiss the danger until we know more. Death may occur sometimes independently or from intercurrent infection.

Treatment.—There is general agreement that complete excision is the only satisfactory method of treatment; but where dense adhesions to vital structures exist such a procedure may be impracticable.

The following is Beye's summary of figures relating to 57 cases operated upon (out of 119 recorded cases):—

Reported Cases	Number of Operations (Complete Excision or Drainage)	Recovered	Improved	Further History Unknown	Died
119	57	22	17	5	12

As these figures include many cases in which complete excision was not attempted, the results cannot be regarded as very unfavourable considering the difficulties of the anatomical situation.

Aurousseau, in an analysis of 38 operations (85 cases), shows the following figures:—

Total Number of Cases	Total of Operations	Incision and Drainage	Incomplete Excision	Complete Excision	Cured	Sequelæ	Died	Further History Unknown
85	38	—	—	—	19	5	11	3
—	—	21	—	—	3	5	10	3
—	—	—	4	—	4	—	—	—
—	—	—	—	13	12	—	1	—

From this it will be seen that with simple incision and drainage a heavy mortality risk is incurred. The risk is much reduced in the case of complete excision; in the case of incomplete excision the figures are too small to warrant any deductions.

Since Beye's article we can trace 5 more cases, including our own. In 4 of these the tumour was successfully removed. In the fifth case, reported by Poynton and Moncrieff, the patient was an infant, and operative measures were impracticable; a very large teratomatous cyst was found at autopsy.

Pathology, etc.—The origin of these cysts is mostly connected with the branchial arches, and is therefore concerned with the fœtal period of life. Beye quotes an apposite statement by Ewing: "The intimate relations of the ectodermal and entodermal layers of the third and fourth arches may explain the variety of epithelium and the connection with the thyroid and thymus,

while the descent of the heart may carry these structures deep into the thorax. Dermoids of the lower mediastinum may result from imperfect closure of the anterior chest wall." Regarding the latter point, Bland-Sutton suggests that they arise from some fault in the median coalescence of the sternum; failure of this kind was noted in our patient, whose sternum was bifid throughout its whole length (*see Fig.* 131).

Ewing divides these tumours into simple dermoids and teratoid tumours. The simple dermoids show epidermal lining with dermal glands, and contain sebaceous material and hair. Teeth may be present. The tumours may be unilocular or multilocular cysts. That is to say, simple dermoids arise from ectoderm; mesodermal and endodermal layers are not represented. "The complex tumours are tridermal, and contain, besides epidermis, bone, cartilage, nervous tissue, intestinal tract, respiratory ciliated epithelium, and thyroid" (Ewing). The same author quotes Ekehorn as pointing out that "the great majority of mediastinal dermoids prove to be tridermal teratomas."

Histological examination of the cyst wall in our case showed an inner lining of squamous epithelium, which in one situation gave place to an exuberant growth of columnar epithelium. Sweat and sebaceous glands, as well as hair follicles, were seen in the subjacent fibrous stroma, as well as some small cysts. There was only scanty evidence of an inflammatory reaction, which is in keeping with the saprophytic nature of the infection present. As the histological findings did not show any mesodermal structures—e.g., muscle and cartilage—the tumour must in our opinion be classed as a dermoid cyst, rather than a tridermal teratoma, in spite of the occurrence of columnar epithelium.

Thus up to date there are records of 124 cases of this condition, and so far as we have been able to ascertain ours is the ninth case where a history of coughing up of hairs has been obtained.

We are indebted to Dr. F. J. Gwynne, of the Radiology Department of the Auckland Hospital, for supplying us with X-ray photographs and his comments thereon.

BIBLIOGRAPHY.

AUROUSSEAU, *Rev. de Chir.*, 1924, 553.
BEYE, *Ann. of Surg.*, 1926, lxxxiii, 577.
BLAND-SUTTON, *Tumours Innocent and Malignant*, London, 1922, 533.
CALDBRICK, *Arch. of Surg.*, 1927, Oct., 660.
CHRISTIAN, *Jour. of Med. Research*, 1902, vii, 54.
EKEHORN, *Arch. f. klin. Chir.*, 1898, lvi, 107.
EWING, *Neoplastic Diseases*, Philadelphia, 1928, 1024.
GRIMM, *Radiology*, 1927, May, 438.
HATHAWAY, *Proc. Roy. Soc. Med.*, (Clin. Sect.), 1923, Dec.
KAVANAGH, *Anæsthesia and Analgesia*, 1927, Aug., 184.
LAMBERT and KNOX, *Trans. Assoc. Amer. Phys.*, 1920, xxxv, 17.
POHL, *Deut. Zeits. f. Chir.*, 1914, cxxx, 481.
POYNTON and MONCRIEFF, *Proc. Roy. Soc. Med.*, 1929, xxii, 635.

CHOLESTEROSIS OF THE GALL-BLADDER: A CLINICAL AND EXPERIMENTAL STUDY.*

By C. F. W. ILLINGWORTH,
CLINICAL TUTOR AT THE ROYAL INFIRMARY, EDINBURGH.

THE lesions characterized by infiltration of the gall-bladder with cholesterol have occasioned considerable interest since, twenty years ago, the 'strawberry' gall-bladder was first described, and their importance surgically has received increasing recognition.

There are two types of cholesterosis. Of these the better known is the 'strawberry' change, in which the infiltration is widespread. Less common is the type in which localized deposits of cholesterol occur in small polypoidal projections of mucous membrane—'cholesterol polypi'. These types appear to have no essential distinction (the difference of appearance being simply due to the massive localization of cholesterol in the second type, leading to prominence and pedunculation of the mucosa), and it is common to find the two types associated.

It is perhaps not generally recognized that cholesterosis of the gall-bladder, far from being a rare lesion, is actually very common. By the courtesy of Professor Wilkie, the writer has had the opportunity of studying 35 cases, of which 21 have been found during the past year, and it is therefore felt that the time is ripe for a survey of all aspects of the disease, clinical and pathological, and for a report upon experimental work which has been carried out in regard to its pathogenesis.

HISTORICAL.

Although the occurrence of cholesterol in the gall-bladder wall appears to have been recognized a long time previously,[1] the macroscopic picture of cholesterosis escaped the attention of surgeons and pathologists alike until as late as 1909—a fact the more remarkable when it is realized that this condition, far from being rare, is found with considerable frequency both at operation and in the post-mortem room, and is, moreover, of very striking appearance unless obscured by discoloration with bile.

The first description of the naked-eye appearance of cholesterosis we owe to Moynihan,[2] who described and illustrated very beautifully three cases in which the condition was found. At that time it was regarded as being due to innumerable small stones embedded in the mucosa, and it was not until several years later that its true significance was recognized.

---

* From the clinic of Professor Wilkie in the Royal Infirmary, Edinburgh, and the Department of Experimental Surgery of Edinburgh University.

MacCarty[3] in the following year described further cases (attributing the appearance to an erosion of the mucous membrane with a secondary bile-staining and fibrosis), and gave the descriptive name of 'strawberry' by which the condition is generally known. More recently Mentzer[4] has applied the term cholesterosis to include all degrees of the disease.

Much of our knowledge of cholesterosis we owe to Boyd,[5] whose masterly paper was published in 1923. In particular, Boyd described in very lucid terms the naked-eye and microscopic appearances, and gave convincing proof of the nature of the lipoid involved in the infiltration.

In recent years the frequency of cholesterosis has led to a somewhat greater interest, especially among French workers, but even yet its importance has not gained widespread recognition.

NAKED-EYE APPEARANCE.

Diffuse Cholesterosis (Strawberry Change).—In a well-marked example of the 'strawberry' gall-bladder the appearance is striking in the extreme (*Figs.* 133–135). The mucous membrane of the whole organ is usually congested and deep red, and scattered over it are innumerable tiny nodular specks of bright-yellow hue, the whole appearance closely resembling that of a ripe strawberry.

FIG. 133.—'Strawberry' gall-bladder. In the congested mucous membrane are many tiny yellow deposits of cholesterol. One larger polypus is also present. (*D. P. D. Wilkie's case.*)

The appearance is even more striking, as Boyd has pointed out, if the gall-bladder is examined under a binocular dissecting microscope. In the healthy organ the surface of the mucosa is seen raised up into numerous tall, thin, gossamer-like ridges (which, from their appearance in cross-section are known, incorrectly, as villi) surrounding deep oval or polygonal hollows. In the 'strawberry' gall-bladder these ridges, instead of being thin and tenuous, are stout and swollen, and within them the lipoid is seen as dense or streaky masses, which, in the words of Boyd, load the villi "much as the delicate birch tree might be weighed down by a load of snow."

The lipoid is for the most part confined to the prominences of the ridges, though in rare cases it may also invade the deeper recesses of the wall. In severe examples practically the whole extent of the summits of the ridges is occupied by the deposits, and they then appear as yellow linear streaks running chiefly in a longitudinal direction.

In other cases the lipoid is deposited, not in streaks, but in pin-head nodules within the villi.

FIG. 134.—'Strawberry' gall-bladder. The cholesterol infiltration of the mucous membrane is well marked and extends over the whole of the gall-bladder. (*Sir H. Stiles's case.*)

The distribution of the lipoid may be widespread, affecting all the ridges throughout the gall-bladder, but is more frequently patchy. Certain portions seem particularly apt to be affected, and in early cases it is not infrequent to find a limited area of cholesterosis at about the mid-point of the gall-bladder or towards the neck, and the rest of the organ may appear healthy. Less commonly the lesion may be found only at the fundus.

A curious and important observation is that, when the lipoid deposits are traced along the ridges towards the ductal end of the gall-bladder, they are found, even in the most marked examples, to end abruptly at a point which in some specimens corresponds to the commencement of the cystic duct; while in others it is placed a little closer to the fundus than this point. It is often found that the infiltrated ridges, which for the most part are arranged longitudinally, merge together at this point into a transverse yellow line; beyond this the mucosa appears perfectly free from lipoid,

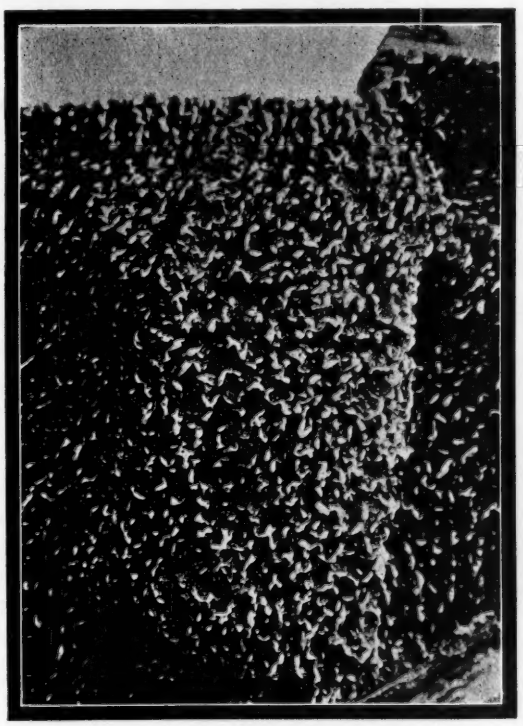


FIG. 135.—The same case as in *Fig.* 134. The ridges of mucous membrane are projecting, distended with lipoid, of pale yellow colour.

or at the most there may be one or two tiny seedlets in an otherwise normal membrane.

Cholesterol Polypi.—Here the lipoid, instead of being scattered diffusely over the gall-bladder wall, is aggregated into larger masses in one, two, or more sites. The villus in which it is deposited becomes progressively swollen and polypoid, and eventually may become a large pedunculated mass attached to the wall by the finest of filamentous stalks (*Fig.* 136). The surface of such a mass may be undulating, gyrate, or lobulated. Its colour, when small, is a bright yellow. Later, as increase in size continues, deposition of bile pigments may give it a greenish or brownish hue. In some cases the polypus consists almost entirely of lipoid material, with a thin epithelial covering which may in part be absent; in others the polypus has a fleshy appearance, with relatively little lipoid. Such polypi may occur singly, or as many as ten or twelve may be present. Not infrequently, in addition, some degree of 'diffuse' cholesterosis is also found.

FIG. 136.—Solitary cholesterol polypus, of large size, attached to the subjacent mucous membrane by a delicate stalk.

Associated Disease of the Gall-bladder. — Cholesterosis may occur either alone or in association with various degrees of inflammation. Occasionally it appears to be a definite pathological entity in a gall-bladder which presents no evidence of any morbid change apart from the cholesterol deposits in its wall. To the naked eye the gall-bladder appears of normal blue colour, quite unthickened, and so transparent that the deposits of cholesterol may be visible from the peritoneal aspect before the gall-bladder is opened; and even on careful histological examination no trace of an inflammatory process is visible. Such cases are, however, uncommon, at least in the examples seen at operation, and only one has been encountered in this series. The majority of cases are associated with some degree of cholecystitis. This may be well marked, the gall-bladder being thickened with old fibrous tissue, but is most characteristically of mild degree. The most common type of gall-bladder to be affected is that which at operation appears just slightly thickened, with some excess of subserous fat and a mild pallor and opacity which mars the healthy blue colour.

Grosser inflammatory disease of the gall-bladder is less common, and this, even when marked, is usually found to be confined principally to the outer

coats, the mucosa being relatively intact. Cholesterosis never occurs in the presence of extensive scarring or atrophy of the mucous membrane, and is never found as a manifestation of acute cholecystitis (though, of course, an acute inflammation may supervene in a gall-bladder which already contains cholesterol deposits).

Cholesterosis may also, rarely, be associated with malignant disease of the gall-bladder.[5] Gall-stones may be present or absent.

The association of cholesterosis with mild rather than severe cholecystitis has been noted especially by Boyd, who found that in one case, where only part of the wall was infiltrated with cholesterol, the remaining parts showed more profound inflammatory change. He concluded from this that the lipoid deposition was an early phenomenon in the disease process and might disappear as the disease advanced.

Table I illustrates the relation of cholesterosis to cholecystitis in the 35 cases of this series. It will be seen that in 11 gall-bladders showing generalized 'strawberry' change, 1 showed no trace of inflammation either to the naked eye or histologically, 1 was only proved to be mildly inflamed by microscopic examination, and 3 others had a very mild degree of chronic cholecystitis.

Similarly, in 10 cases out of 20 gall-bladders with patchy 'strawberry' cholesterosis, the associated inflammatory change was meagre, and the same was true for all the 4 cases in which polypi alone were found.

In general, the gall-bladders containing stones were considerably more thick-walled than the others, and if these cases are excluded the preponderance of mild inflammatory changes is still further increased, for moderate or gross chronic cholecystitis occurred in only 4 of the 18 cases without stones.

Table I.—Relation of Cholesterosis to Cholecystitis.

Type of Cholesterosis	No. of Cases	Grade of Chronic Cholecystitis					
		None	Recognized only microscopically	Slight opacity of wall	Wall moderately thickened	Wall grossly thickened	Acute exacerbation
* Generalized 'strawberry' ..	11	1	1	3	4	1	1
† Patchy 'strawberry' ..	20	0	1	9	3	6	1
Polypi alone	4	0	2	2	0	0	0
Total	35	1	4	14	7	7	2
Total cases with stones ..	17	0	0	5	4	6	2
Total cases without stones ..	18	1	4	9	3	1	0

* One of these also contained polypi. † Four of these also contained polypi.

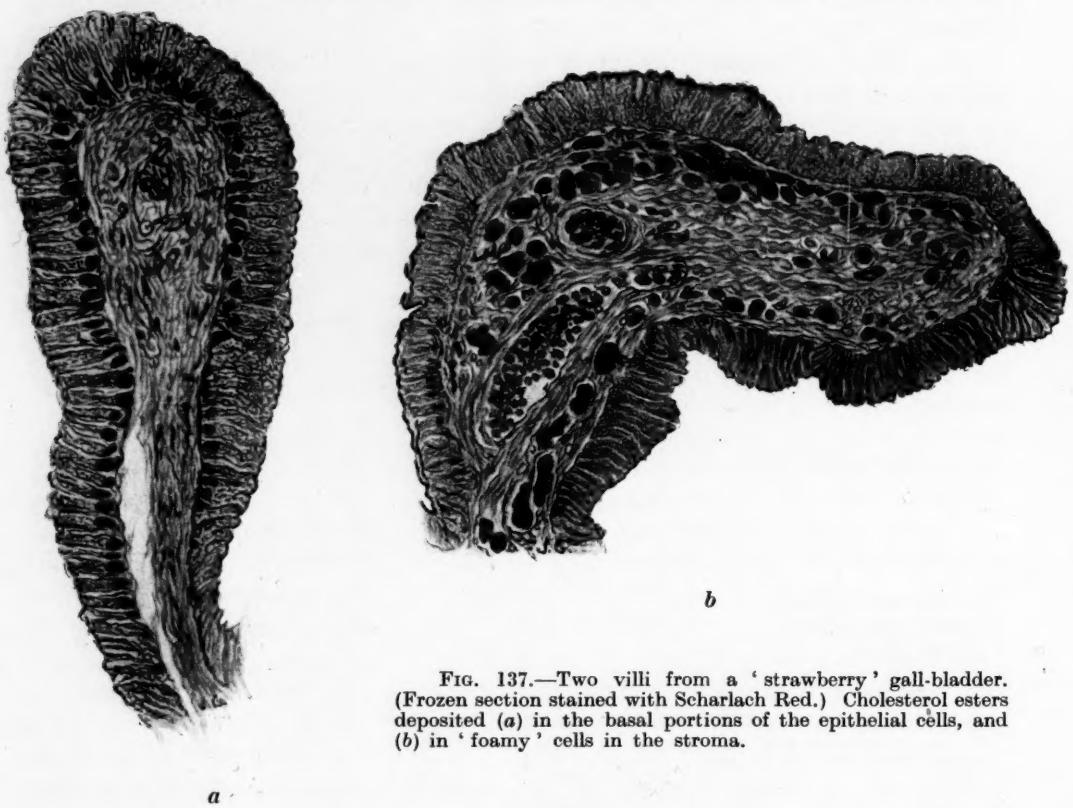
Occurrence of Gall-stones with Cholesterosis.—Gall-stones have been present in nearly half of the cases in this series, namely in 17 of the 35 cases of cholesterosis.

It is of particular interest to note that in the majority of these cases the stones have been composed of pure or almost pure cholesterol, either single stones (cholesterol 'solitaires') or multiple (of 'mulberry' type), and a relationship between the deposit of cholesterol in the mucous membrane and the formation of cholesterol stones seems clearly demonstrated.

MICROSCOPIC APPEARANCE.

General Histological Characters.—It has already been stated that cholesterosis may be associated with divers changes in the gall-bladder wall, and the histological appearance is therefore equally varied. There may be an extensive fibrosis of the deeper parts of the wall, or a less chronic lesion with engorgement of the vessels and some round-celled infiltration, or, on the other hand, the gall-bladder may show no deviation from the normal except for the deposit of lipoids.

In the majority of cases, however, certain characteristic features will be noted. However marked and old-standing the inflammatory changes, they



a *b*

FIG. 137.—Two villi from a 'strawberry' gall-bladder. (Frozen section stained with Scharlach Red.) Cholesterol esters deposited (*a*) in the basal portions of the epithelial cells, and (*b*) in 'foamy' cells in the stroma.

are mainly confined to the outer coats of the wall. The mucous membrane usually shows some hyperplasia, sometimes well marked; the villi are enlarged, elongated, and almost pedunculated. The epithelial layer, which is usually complete unless eroded by pressure of stones, may be thrown into numerous

folds which, in sections, sometimes give a false impression of glandular acini. The stroma of the mucous membrane also shares in the hyperplasia. The connective-tissue cells are proliferated, and in addition there is often a mild degree of infiltration with leucocytes. Giant cells of the 'foreign body' type, which occur around many cholesterol deposits, are never seen in the 'strawberry' gall-bladder. A characteristic feature in the stroma is the vascular dilatation, which may be well marked.

Situation of the Lipoid.—The main mass of the lipoid is deposited in the mucous coat, though occasionally in advanced cases traces may also be visible in the fibromuscular layer. In the mucosa the deposits may be either in the epithelium or in the stroma. In some specimens the whole of the lipoid is in the one situation, in others it is in both; sometimes in the same gall-bladder one portion of the section shows the epithelial deposit, another that in the stroma (*Fig.* 137). Moreover, different portions of the epithelial cells or of the stroma may be affected in different cases, giving rise to variations of appearance which are not readily explicable.

In the Epithelial Cells the most characteristic infiltration of lipoid is at the bases of the cells, in the form of large fatty globules which stain a bright red with Scharlach R. (*Fig.* 137 *a*). These globules are usually localized to the tips of the villi, but in marked cases they may extend to the intervening depressions, forming a sort of scarlet border to the whole section. In this basal situation the lipoid consists mainly of esters of cholesterol.

Less commonly the superficial part of the epithelial cells contains lipoid deposits, in the form of multiple fine granules, which contain little or no cholesterol and appear to consist of unsaturated fatty acids (*Fig.* 138).

FIG. 138.—Section of 'strawberry' gall-bladder (stained osmic acid). Note the large grey 'foamy' cells (containing lipoid) in the stroma, and the dense black lipoid in the epithelium.

In the Stroma of the Mucous Membrane the lipoid occurs characteristically in large amount. Though extracellular deposits have been described, these must be rare, and in all the cases of this series the lipoid has been situated inside cells of various types.

The most important lipoid-containing cell is one which, from its appearance in paraffin sections, has been called 'foamy' (*Fig.* 138). It is a large mononuclear endothelial cell with a small, dark-stained nucleus and a very delicate reticular protoplasm, in the meshes of which are contained numerous minute droplets of cholesterol esters. Such foamy cells occur at first near the tips of the villi as small rosettes or in linear arrangement (*Fig.* 137 *b*). Later they may collect in large numbers, distending the villi to resemble air

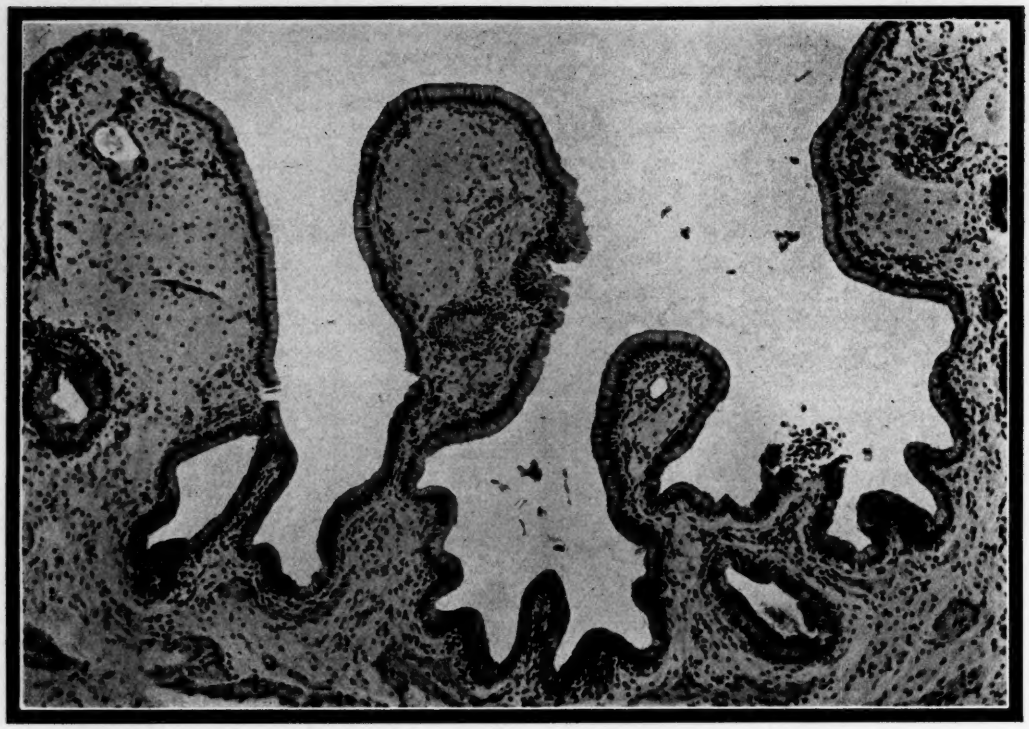


FIG. 139.—Paraffin section from 'strawberry' gall-bladder (stained hæmatoxylin and eosin). The villi of the mucous membrane are swollen by numerous foamy lipoid-containing cells. These are seen as pale areas, from which the lipoids have been dissolved during preparation of the section. (× 100.)

balloons (*Fig.* 139) and penetrating down into the deeper parts of the stroma (*Fig.* 140). It is to the mass of lipoid in these foamy cells that the yellow colour of the 'strawberry' gall-bladder is mainly due.

FIG. 140.—Frozen section from a 'strawberry' gall-bladder (stained with Scharlach Red). Note the dense masses of cholesterol distending the two villi. (× 50.)

Lipoid may also be found as minute droplets in small elongated or polygonal cells of the stroma, and, less commonly, it may occur in the endothelial cells of blood-vessels. In these two situations it contains no cholesterol.

The occurrence of lipoid has been noted in the lumen of blood-vessels, though this must be a rare phenomenon. Boyd described one case, in which complete proof was lacking, and Bergeret and Dumont[6] have described another. In this latter case actual foamy cells were seen in the lumen of a venule.

NATURE OF THE LIPOIDS.

Boyd has described with great lucidity the methods in use for the recognition of lipoids, and they require but a brief outline here. It will be recalled that cholesterol and its esters can be most readily distinguished by their property of rotating the plane of polarized light, a property not possessed by other lipoids commonly occurring in the body. Cholesterol can be distinguished from its esters by various staining reactions, and, more exactly, by their melting points, that of pure cholesterol being 148° to 150° C. as compared with 37° to 42° C. for the oleate. Under the polarizing microscope a section of a 'strawberry' gall-bladder presents a brilliant appearance, large masses of anisotropic lipoid standing out in illumination against the dark background of the rest of the tissue. An interesting phenomenon is observed if the section is heated to approximately 40° C. At this point the lipoid masses disappear and their place is taken by numerous large and small 'Maltese crosses' of brilliant white colour, an appearance characteristic of the 'fluid crystalline state' in which the lipoid is physically fluid yet retains the optical properties of the solid.

Boyd has shown that much of the lipoid of the 'strawberry' gall-bladder responds to the tests for esters of cholesterol,* but it must be remarked that this applies only to the lipoid in certain situations (in the 'foamy' cells and in the base of the epithelium). In other parts of the mucosa non-cholesterol lipoid is found, which responds to the tests for unsaturated fatty acids. Thus it is seen that the 'strawberry' gall-bladder is really an infiltration with many complex lipoids, of which combinations of cholesterol and fatty acids are merely one manifestation, though no doubt the most important.†

BACTERIOLOGY.

Whenever possible cultures in Rosenow's brain-glucose-broth medium, or in ordinary glucose broth, have been taken from the gall-bladder wall, the bile, and the cystic lymphatic gland, and in a few cases also from the stones present. In the great majority of cases these tissues have proved sterile, and in the rest no characteristic flora has been found (*Table II*).

Table II.—Bacteriology of Cholesterosis.

Tissue	No. of Cases	Sterile	Infected	Streptococci	B. Coli	Other Organisms
Gall-bladder wall ..	22	16	6	3	2	1
Bile	23	19	4	2	1	1
Cystic gland ..	21	19	2	1	0	1
Stone	3	2	1	1	0	0

* Strictly speaking, the tests mentioned apply to mixtures or loose combinations of cholesterol and fatty acids as well as to true esters, and it is probable that in cholesterosis various such mixtures occur. For the sake of simplicity they are here referred to as true esters.

† Since the completion of this paper a case has been observed of a well-marked 'strawberry' gall-bladder in which large masses of lipoid filled the epithelial cells and traces occurred in the stroma. In neither situation was any trace of doubly refractile cholesterol present, all the fat giving the reactions for fatty acids.

CHARACTER OF THE BILE IN CHOLESTEROSIS.

As Moynihan noted in his original paper, the bile is frequently dark, tarry, and extremely concentrated. This is especially the case where the cholesterosis is well marked (*Table III*), and of the 7 gall-bladders with generalized 'strawberry' change in which the concentration of the bile was noted, all contained tarry bile. Apart from being highly concentrated, however, the bile is quite clear, and it is unusual to find any trace of turbidity.

Table III.—Character of the Bile in Cholesterosis.

Cholesterosis	No. of Cases	Clear Bile			Turbid Bile
		Dilute	Tarry	Tarry, with cholesterol flakes	
Generalized 'strawberry' ..	7	0	6	1	0
Patchy 'strawberry'	15	6	7	0	2
Polypi	2	0	2	0	0

It is true that the condition of the bile in a patient prepared for operation does not accurately represent that usually present, for in the fasting individual rapid concentration takes place; but comparing the bile with that of other patients similarly treated there seems no doubt that undue concentration is the rule.

The Cholesterol Content of the Bile, like its concentration, appears to be increased in cholesterosis. *Table IV* shows the readings obtained in six cases of this series. It will be seen that there is a very definite increase in the content as compared with the controls, especially where the cholesterosis is marked in extent.

Table IV.—Cholesterol Content of Gall-bladder Bile.

Gall-bladder	Cases	Cholesterol Content	Averages
		Mgrm. per cent	
Patchy 'strawberry'	1	466	503·6
	2	445	
	3	600	
Generalized 'strawberry'	1	636	969·6
	2	1033	
	3	1240	
Controls (no cholesterosis)	1	156	329·3
	2	290	
	3	542	

These observations are of interest from two points of view: (1) The high index may account for the frequent presence of stones of pure cholesterol, the result of precipitation from the bile; (2) As will be considered later, a high cholesterol content may be important as one of the factors leading to cholesterosis.

CLINICAL FEATURES OF CHOLESTEROSIS.

ETIOLOGY.

If surgically removed gall-bladders are carefully examined in the fresh state, a degree of cholesterosis visible to the naked eye is found in a considerable proportion of cases, and well-marked typical 'strawberry' change is by no means rare. In 100 consecutive cholecystectomies during the past twelve months cholesterosis in some form was present in 21, a frequency corresponding fairly closely with that of other observers. In a larger collection of 35 cases of all types of cholesterosis, the well-marked 'strawberry' type was present in 11, and the patchy 'strawberry' type in 20. Cholesterol polypi occurred alone in 4 cases, and were also present in 5 of those showing the 'strawberry' change.

Sex.—Cholesterosis appears to affect the sexes equally. In the present series men were affected in 4 (or 20 per cent) of 20 consecutive cholecystectomies; women in 17 (or 21·25 per cent) of 80. These figures, being based upon surgical cases in which women preponderate, do not necessarily represent the total incidence in the two series, but it is interesting to note that in a large autopsy series reported by Mentzer[4] a similar close ratio was noted.

Age.—Cholesterosis is a condition chiefly of middle life, though it has been found at the early age of 13. Mentzer states that the average age is 35. In this series the youngest patient was aged 28, the eldest 60, and the great majority exceeded 45 years.

Social State.—Remarkable similarity has been noted in the incidence of the disease in hospital and in private cases. Cholesterosis occurred in 12 (or 21 per cent) of 57 consecutive cholecystectomies in hospital, as compared with 9 (or 20·9 per cent) of 43 private cases.

ASSOCIATED LESIONS IN OTHER ORGANS.

Excluding cholecystitis and gall-stones, cholesterosis seems to have no relationship with other intra-abdominal disease. In 35 cases duodenal ulcer co-existed twice, hydronephrosis once, and pronounced visceroptosis once. Appendicectomy was performed in a considerable number of cases, but usually as a routine measure, and gross chronic appendicitis was present in only 2 cases.

SYMPTOMATOLOGY.

The symptoms presented by patients with cholesterosis of the gall-bladder are extremely varied, and even where other conditions such as gall-stones or marked cholecystitis are absent, the symptoms may be either very severe or entirely lacking.

Other workers have remarked that in cholesterosis the symptoms may be of some severity. Thus, Chiray and Pavel[8] state that the condition is a painful one, sometimes extremely so, and they describe cases in which the predominating feature was that of severe colicky pains recurring at frequent intervals. Fever, they state, is rarely absent at some stage of the disease, and icterus may be observed.

A history of this type has been obtained in four cases of uncomplicated cholesterosis in this series, with the exception that none of them has given

evidence of attacks of fever. All of them had suffered from attacks of pain, which appears to have been intense. One patient volunteered that the pain was worse than that of childbirth, and another stated that it made her roll about and perspire freely. The pain in all these cases closely resembled that of biliary colic, striking the patient in the right hypochondriac region and radiating to the scapula or to one or other shoulder, and necessitating the administration of morphia. In two cases the pain was followed by definite jaundice of several days' duration. In the intervals between the attacks these patients suffered from symptoms like those of chronic cholecystitis, aching pain in the hypochondrium, soreness of the skin in this region, flatulence, and abdominal distension.

In spite of these clearly defined histories of severe symptoms, the gall-bladders in these four cases showed extremely little change except for the presence of gross deposits of cholesterol. No stones were present, and the degree of cholecystitis was very limited. In one case there was absolutely no histological trace of an inflammatory lesion; in a second such evidence was only obtained on microscopic examination, and in the remaining two the cholecystitis was of a very mild degree. Yet in all four cases the absence of any other gross intra-abdominal lesion and the curative effect of cholecystectomy prove that the symptoms had originated in the biliary condition.

The following is a typical case of this group:—

Case 2829.—Mrs. T., age 34, multiparous.

HISTORY.—During the two years previous to admission the patient had had pain on the right side of the abdomen. At first it came in attacks of great severity, which doubled her up and were 'worse than labour pains'. The pain was felt just below the right costal margin and passed to the back below the right scapula. She often had also a pain in the right shoulder. Most of the attacks came on during the day. They might last for a few hours and then pass off, or might recur at intervals over a few days. The pain came is spasms.

Ever since the attacks commenced she had complained of a soreness in the right side, which prevented her wearing tight clothing. She had occasionally been a little yellow-coloured, but definite jaundice had not been noticed. She had no indigestion and could eat any kind of food. Food never relieved the pain. Vomiting occurred when the pain was severe.

OPERATION.—Stomach, duodenum, common duct, and other viscera appeared healthy. The gall-bladder was for the most part of healthy appearance, but seemed a little pale and opaque at the fundus. It was removed. It contained thick, tarry bile, and no stones. The mucosa had the typical appearance of a 'strawberry' gall-bladder, with deposits of cholesterol in linear streaks over a somewhat congested mucous membrane. The wall of the gall-bladder was only very little thickened, and microscopic examination revealed only mild cholecystitis.

PROGRESS.—Reported nine months later: no recurrence of symptoms and is now well and symptom-free.

Such cases with severe symptoms are, however, rare, and the majority of patients give merely a history indicative of chronic cholecystitis—that is to say, the deposit of cholesterol does not appear to aggravate the symptoms due to the accompanying inflammatory change.

In others, again, the disease may be completely symptomless. Such was the case in a patient, age 31, whose only symptoms were those of stiffness in the hip-joint and pains in this region. No signs of local disease of the joint could be found, and as routine examination revealed slight tenderness over

the gall-bladder, this organ was removed as a probable focus of infection—a procedure which resulted in complete disappearance of the pain and joint stiffness. The gall-bladder proved to have a definite, though mild, degree of inflammation, and showed well-marked 'strawberry' change.

CLINICAL SIGNS.

These give little help in the diagnosis of cholesterosis. The general appearance of the patient is in no way dissimilar from that usually associated with cholecystitis, and the patients are usually, but not invariably, well nourished or stout. Local signs are merely those of chronic cholecystitis.

SPECIAL EXAMINATIONS.

Cholecystography.—The appearance of the cholecystogram depends upon the degree of accompanying cholecystitis or upon the presence of stones. If moderate cholecystitis coexists or stones are present, obscurity or absence of the cholecystographic shadow will result; but, on the other hand, if cholesterosis is accompanied by little inflammatory change, the cholecystographic examination will show no deviation from the normal. Thus, in 8 cases of this series in which deposition of cholesterol was the main change visible to the naked eye, the administration of sodium tetraiodophenolphthalein invariably resulted in a dense 'normal' shadow of the gall-bladder, which, moreover, diminished markedly in size following a fat meal.

This examination (which incidentally indicates that cholesterosis *per se* does not interfere either with the concentrating function of the gall-bladder or with its contractility) is therefore only of value in the diagnosis of cholesterosis in that it may exclude the grosser diseases of the gall-bladder.

Blood-cholesterol Examination. — The cholesterol content of the blood has such a wide 'normal' range of values, and is subject to abnormal increase from such varied pathological or even physiological conditions (e.g., diabetes, parenchymatous nephritis, xanthoma, pregnancy), that the value of this estimation as a diagnostic measure must be limited. The recorded cases of cholesterosis in which blood-cholesterol estimations have been carried out are

Table V.—Blood-cholesterol Estimations.

Gall-bladder	No. of Cases	Blood-cholesterol			
		Low	Low normal	High normal	Raised index
Generalized 'strawberry' ..	6	0	2	1	3
Patchy 'strawberry'	3	0	1	2	0
Controls: Cholecystitis No cholesterosis	14	4	2	4	4

only two in number, but, in spite of this lack of concrete data, a state of hypercholesterolæmia has been presumed by many writers to be of constant occurrence in cholesterosis. It appears, however, that this is incorrect. In the present series estimations of the blood-cholesterol index have been carried out upon 9 cases of cholesterosis of all grades, and, as a control series, upon 14 cases

operated upon for lesions of the gall-bladder other than cholesterosis. Reference to *Table V* will show that an increased reading was almost as frequently obtained in the control series as in the cases with cholesterosis, and that exactly two-thirds of the latter cases were within the limits of normal.

The blood-cholesterol estimation cannot therefore be regarded as of practical value in the diagnosis of cholesterosis.

DIAGNOSIS.

It will be obvious from the varied symptomatology that the diagnosis of cholesterosis must present great difficulties. On the one hand, severe pain simulating biliary colic and followed by transient jaundice will lead to the suspicion that a stone is present in the gall-bladder, and, on the other, the symptoms may be so trifling as to be missed.

In the majority of cases, however, the symptoms and signs point clearly to some lesion of the biliary tract, and the diagnosis of cholesterosis must depend either upon the exclusion of grosser lesions or upon special methods of examination. Of these, the one which seems to offer most assistance in the diagnosis is cholecystography.

It is now generally recognized that cholecystography, though by no means an infallible diagnostic measure, does give an indication either of gross chronic cholecystitis or of the presence of all stones except the smallest, and a 'normal' cholecystographic shadow excludes these conditions. In a patient with a clear biliary history and a 'normal' shadow the diagnosis is therefore narrowed down to one of the following lesions: mild chronic cholecystitis, cholesterosis, or small calculi. It must be admitted, however, that in the present state of our knowledge the diagnosis of this condition can only be tentative.

TREATMENT.

As cholesterosis is rarely recognized until an extirpated gall-bladder has been opened, consideration of the treatment is of academic rather than practical interest. In two circumstances, however, cholesterosis may be recognized at an earlier stage. The yellow deposits in the mucous membrane may be discovered when the gall-bladder is opened for the purpose of cholecystostomy, or occasionally they may be recognized in the course of an exploratory laparotomy, when they may be visible through the thin translucent gall-bladder wall.

In the former circumstances the indication for treatment seems clear. The gall-bladder having already been opened, only two courses are available—either drainage or extirpation. Drainage can have no therapeutic effect upon a disease localized to the wall of the gall-bladder, and is more likely to result in increased traumatization, infection, and scarring of the mucosa. Cholecystectomy is therefore clearly desirable.

The indication in the second circumstance, where cholesterosis is recognized in an untouched gall-bladder, is not so clear, and must be based upon our knowledge of the etiology of cholesterosis, and upon its symptoms and the possibility of spontaneous cure. In regard to this possibility little is at present known, and it is conceivable that in the absence of inflammatory lesions cholesterol deposits may eventually undergo absorption, with complete

restitution to the normal. The observations of Boyd and others, however, indicate that the disappearance of cholesterol is usually associated with an extension of the inflammatory process already present, and the treatment should therefore be directed towards the prevention of this. Medical measures are not known to have any influence upon lesions of the gall-bladder wall, and if symptoms are present which point to the biliary tract, in the absence of contra-indications the operation of cholecystectomy is called for. This treatment has been carried out in all the cases of this series, and, though the period since operation is in most instances too short to justify a final opinion, the results so far seem eminently satisfactory. In 6 cases of uncomplicated cholesterosis which can be traced, the period since operation has varied from six months up to four years These 6 cases include the 4 patients who gave histories of severe symptoms, and all six state that since the operation no recurrence of these symptoms has taken place.

PATHOGENESIS OF CHOLESTEROSIS.

INFILTRATION OR DEGENERATION ?

Throughout this paper cholesterosis of the gall-bladder has been referred to as an *infiltration*, and this description of its pathological significance requires some support. Can cholesterosis be classified as an infiltration of certain cells by cholesterol entering them from without, or may it be the result of a degeneration, i.e., an unmasking of cholesterol previously present in the cells combined in invisible form ?

The whole appearance of the 'strawberry' gall-bladder, with a vast amount of lipoid in a stroma normally scanty and relatively acellular, is strongly suggestive of an infiltration, and this has been quite definitely proved to be the case by Boyd,[5] who showed that whereas the cholesterol content of the wall of normal gall-bladders, estimated by colorimetric methods after extraction in chloroform, ranged from 0·5 to 1·7 per cent of the dry weight, and that of an inflamed, non-cholesterol gall-bladder was 0·36 per cent, the corresponding estimations for 'strawberry' gall-bladders reached the immense figures of from 34·6 to 60·5 per cent.

General Considerations.—Great controversy has raged, especially in French literature, over the relation of cholesterol infiltration to cholecystitis. Gosset[7] and his associates maintain that cholesterosis represents an aseptic process in stone formation. Chiray and Pavel,[8] Lecène and Moulonguet,[9] and others equally insist upon an inflammatory basis, accounting for the cases in which histological evidence is lacking by the assumption that a former cholecystitis has undergone complete resolution. Regarding cholesterosis as a non-inflammatory change, Mentzer has endeavoured to relate it to a general change in lipoid metabolism, and Stewart,[10] amongst others, has regarded it as a result of hypercholesterolæmia.

From the observations which have already been described it must be evident that hypercholesterolæmia is too inconstantly found to be regarded as essential, and the occasional absence of cholecystitis cannot be disregarded, in spite of the view quoted above. Moreover, infection and hypercholesterolæmia together do not necessarily result in cholesterosis (*see* the controls in

Table V), and clearly other factors must be sought for. It is believed that these may be found in a consideration of the especial relation of the biliary tract to cholesterol metabolism.

The bile is, except for the milk during lactation, by far the most important vehicle for the excretion of cholesterol, and its cholesterol content, especially after concentration in the gall-bladder, may be high. What effect the gall-bladder exerts upon the bile-cholesterol awaits proof. Naunyn[11] has always maintained that the gall-bladder excretes cholesterol into the bile, whereas others believe that it reabsorbs some of the cholesterol into the blood-stream. Whichever view is right, it is believed that some dysfunction of the gall-bladder is intimately concerned in the production of cholesterosis; hence the strict localization of the deposits to the gall-bladder and their absence from the ducts. Before these various features are considered more fully, it will be of advantage to study certain cholesterol infiltrations in other sites in the body.

Other Cholesterol Infiltrations.—Pathological infiltrations with cholesterol, which may occur in diverse situations throughout the body, have been classified by Stewart[10] into two principal categories, according as they result from local tissue changes or from a general increase in the cholesterol content of the body. In many of these infiltrations the microscopic appearance has much in common with that of the 'strawberry' gall-bladder, and the foamy cells in particular are frequently met with. The cholesterol present is usually in the form of the ester, often admixed with other lipoids. It is usually intracellular, but may be deposited in the extracellular tissues, in which case a foreign-body reaction of the giant-celled type may occur. Occasionally the cholesterol occurs in an invisible form, in which it neither stains with fatty stains nor exhibits any optical activity, and can only be demonstrated by chemical analytical methods, being combined, in all probability with proteins, in a masked form.[12]

Certain of these infiltrations deserve especial mention, as they throw some light upon the etiology of cholesterosis.

Cutaneous Xanthoma.—The yellow cutaneous or subcutaneous nodules of this disease are characterized by the presence of foamy cells which closely resemble, both in form and lipoid content, the cells of the 'strawberry' gall-bladder. In regard to pathogenesis, the important feature is the almost invariable finding of a raised blood-cholesterol content, and this is usually regarded as the essential etiological factor. A second, localizing, factor is suggested in some cases, as where xanthoma followed a mosquito bite,[13] and this is supported by the experiments of Anitschkow,[14] who produced xanthomatous lesions in hypercholesterolæmic animals by the subcutaneous injection of aseptic irritants.

Xanthosis of the Fallopian Tubes.—This is a rare finding in cases of chronic salpingitis or pyosalpinx. Pick,[15] and Daniel and Babès[16], have described cases in which cholesterol deposits occurred closely resembling those in the gall-bladder in naked-eye and microscopic appearance. In some cases the cholesterol was distributed uniformly in the mucosa; in others it formed numerous yellow polypi. In these cases there is no evidence of a raised blood-cholesterol index, and the *local* excess of cholesterol in the diseased adnexa has been regarded as of importance.

RELATION OF HYPERCHOLESTEROLÆMIA TO CHOLESTEROSIS.

It has already been indicated that on clinical grounds there is little evidence that a raised blood-cholesterol index can cause cholesterosis. In two-thirds of the cases in which it was estimated the index was normal, and, conversely, it is known that the blood-cholesterol index may reach such immense figures as 1250 mgrm. per cent,[12] yet fail to give rise to cholesterosis.

Experimentally, Blaisdell and Chandler[17] have upheld the view that hypercholesterolæmia *alone* may cause cholesterosis, for in rabbits they have shown that a prolonged course of feeding with cholesterol leads to the deposit of this substance in the gall-bladder wall. This finding, which I have repeated and confirmed, cannot, however, be accurately compared with cholesterosis in the human being, for in animals, if feeding with cholesterol is maintained, as in the experiments referred to, for a prolonged period, cholesterol is deposited in large amounts in the liver, kidney, spleen, adrenals, and other organs, and gives rise to gross atheroma of the aorta and other vessels—lesions which quite overshadow a rather scanty microscopical deposit in the gall-bladder wall.

The following is an extract from one of a series of experiments which illustrate this point:—

Experiment 1.—Cholesterol was administered to four healthy rabbits over periods from 11 to 15 weeks, each rabbit receiving daily, in addition to its usual portion of bran and greenstuff, 0·2 grm. of pure cholesterol, which was administered, for convenience, suspended in butter.

During the period of feeding one of the animals was pregnant on two occasions. The blood-cholesterol index in all cases was very markedly raised to many times its normal value.

At the end of the period the animals were killed. All showed gross deposits of cholesterol in the wall of the aorta and in many other organs, but in only two was there any trace of cholesterol visible in the gall-bladder wall. In these two cases minute droplets were visible, chiefly in the subserous coat of the gall-bladder and to a less extent in the mucosa, but this scanty deposit contrasted markedly with the appearance of the adjacent liver, in which large masses of doubly refractile material were visible.

In one animal at the end of three months the blood-cholesterol content had been raised from 105 mgrm. per cent up to the immense figure of 1136 mgrm. per cent. Excessive deposits of cholesterol esters were present in both kidneys, which were typical gross examples of 'myelin kidney'; there was a considerable degree of aortic atheroma, and large deposits of cholesterol were present in the adrenals, ovaries, liver, spleen, and other organs; yet the gall-bladder appeared perfectly healthy to the naked eye, and, microscopically, contained only minute traces of cholesterol.

RELATION OF INFECTION OF THE GALL-BLADDER TO CHOLESTEROSIS.

Diametrically opposed views are held in regard to the importance of the infective factor in cholesterosis. Whilst it is generally conceded that in the large majority of cases a greater or less degree of cholecystitis is present, occasional cases have been reported in which no histological evidence of infection is found, and in view of the fact that most papers on the 'strawberry' gall-bladder are based upon surgical cases which are usually brought to operation on account of an accompanying inflammation, these exceptional non-inflammatory cases are of particular importance.

Gosset and his associates have noted the absence of infection in certain of their cases, and Mentzer from autopsy studies confirms this. In the present series several cases have occurred in which little evidence of inflammation appeared to the naked eye, but in all of these with one exception the microscopic examination gave undoubted evidence of mild cholecystitis. In one gall-bladder, however, although a marked degree of lipoid infiltration was present, no trace of an inflammatory lesion could be found either by histological or bacteriological examination.

In their staunch advocacy of an infectious origin of cholesterosis, Chiray and Pavel have disregarded all histological evidence to the contrary, believing that a mild cholecystitis may disappear, leaving no recognizable trace behind it except the deposits of cholesterol to which it has given rise; but with our present knowledge of the diseases of the gall-bladder this view seems unsupported. Judging by our present criteria, therefore, it must be admitted that an infection is not essential, though undoubtedly of frequent occurrence, with cholesterosis.

Factor of Biliary Stasis.—It has already been stated that in most cases of cholesterosis the bile is dark and tarry, and this has been taken by some writers as evidence of undue stasis in the gall-bladder. This contention cannot, however, be supported. The concentration of the gall-bladder bile depends not only upon the amount of stasis which exists, but also on two other factors—namely, the bile concentration when excreted from the liver, and the absorptive power of the gall-bladder wall. Clinically, cholecystographic examination of several of the patients of this series showed that the gall-bladder decreased greatly in size after a fat meal, in some of the cases becoming almost emptied, and it may therefore be concluded definitely that biliary stasis has no part in the production of cholesterosis.

EXPERIMENTAL PRODUCTION OF CHOLESTEROSIS BY HYPERCHOLESTEROLÆMIA PLUS INFECTION OF THE GALL-BLADDER.

It was felt that the importance of the factors of hypercholesterolæmia and infection could be most satisfactorily demonstrated by animal experimentation, and, as either of these factors acting alone fails to give rise to gross lipoid infiltration of the gall-bladder, a combination of the two was next attempted: this proved completely successful.

The method employed to produce a chronic cholecystitis was the inoculation of avirulent organisms directly into the wall of the gall-bladder. It is well recognized that in a considerable proportion of chronically inflamed gall-bladders cultures taken from the wall will yield a growth of streptococci which are non-hæmolytic, slow-growing, and avirulent; and A. L. Wilkie[18] has shown that if these organisms are injected between the layers of the gall-bladder wall, no acute inflammation occurs, but a slowly progressive chronic cholecystitis appears, at first localized to the site of inoculation, but later spreading over the whole gall-bladder. The whole process resembles that seen clinically in cases of mild or moderate chronic cholecystitis, in which inflammatory changes are chiefly localized to the outer coats of the wall, the mucosa remaining relatively intact.

The procedure adopted was as in the following experiment:—

Experiment 2.—A healthy male chinchilla rabbit was fed with 0·2 grm. of pure cholesterol in butter daily, along with its ordinary ration of bran and greenstuff, for

a period of 13 weeks. One week after the commencement of the feeding laparotomy was performed. The gall-bladder appeared healthy. With a syringe and fine needle an intramural injection of a saline suspension of streptococci was then made into the inferior wall of the gall-bladder at its mid-point. The streptococcus was one which had been isolated several months previously from a case of cholecystitis, and belonged to the *viridans* group. For the inoculation the growth from a 24-hour

FIG. 141.—Experimental production of 'strawberry' gall-bladder. In a rabbit, fed with cholesterol to raise the blood-cholesterol content, a mild chronic cholecystitis was produced by injecting avirulent streptococci into the gall-bladder wall. A typical 'strawberry' gall-bladder resulted, clearly recognizable to the naked eye. This section (stained with Sudan IV) shows large masses of cholesterol in the epithelium and stroma.

agar slope culture was suspended in 10 c.c. of saline solution, and about one minim was injected.

Thirteen weeks after the commencement of the experiment the rabbit was killed. At this time it was extremely well nourished and appeared healthy. The abdomen was free from adhesions, the scar of the operation wound being barely discernible. The gall-bladder was of average size; its inferior surface had an opaque, pearly-white appearance, and on palpation seemed thickened. Its deep

wall, when separated from the liver, appeared relatively normal. The other viscera showed relatively little change to the naked eye, though microscopically deposits of lipoid were visible in the liver and adrenal glands.

The gall-bladder contained a large semi-solid concretion and a few drops of turbid bile. The amount present was insufficient for an accurate estimation, but rough tests showed the presence of cholesterol in excess.

The inner aspect of the gall-bladder presented a striking appearance. Through its whole extent the mucosa was raised up into linear ridges of a bright yellowish-white colour, the whole appearance being very like that of a clinical 'strawberry' gall-bladder, and this similarity was further enhanced by the fact that close up to the commencement of the cystic duct the infiltration stopped completely, the cystic and common ducts being to all appearances free from lipoid.

In frozen sections from the gall-bladder (*Fig.* 141) the similarity to clinical cholesterosis was completely demonstrated, for the lipoid was found in the two regions where it would be expected—namely, in the stroma and the epithelial cells of the mucous membrane—and much of the lipoid conformed in physical and chemical characteristics with an ester of cholesterol; i.e., it was anisotropic, and it melted, with the production of Maltese crosses, at from 37° to 42° C.

FIG. 142.—Experimentally produced 'strawberry' gall-bladder. Paraffin section of gall-bladder seen in *Fig.* 141 (stained hæmatoxylin and eosin). Foamy cells in stroma give an appearance exactly like that of typical 'strawberry' gall-bladders in man. (× 150.)

Paraffin sections confirmed the similarity, for, in addition to numerous small cells of inflammatory origin, the stroma of the mucosa contained a number of typical 'foamy' cells, which were most marked in the rather prominent villi, but also extended into the deeper part of the stroma (*Fig.* 142). In sections stained with osmic acid these 'foamy' cells were seen to contain lipoid granules stained a faint grey.

In another rabbit, similarly treated except that the streptococcal inoculation was made only 4 weeks before the animal was killed, the appearance of the gall-bladder was even more striking. This organ, which was small and somewhat thickened, contained a few drops of fluid bile and a large soft yellow mass, which was taken at first to be thick pus adherent to the mucosa. More careful examination showed, however, that this was incorrect, and in microscopic sections it was seen that the whole of this mass was composed of large 'foamy'-celled areas situated within the mucosa, the condition being that of a very much exaggerated 'strawberry' change.

It will be seen, then, that cholesterosis of the gall-bladder can be readily produced experimentally by inducing an inflammation of the gall-bladder in a hypercholesterolæmic animal. It remains to be shown how this experimental finding can be correlated with the clinical observation that either cholecystitis or hypercholesterolæmia may be lacking. It is believed that this correlation can best be effected by a study of the function of the gall-bladder in regard to cholesterol.

FUNCTION OF THE GALL-BLADDER IN REGARD TO CHOLESTEROL.

Two opposing views are held in regard to this relationship, namely: (1) That the gall-bladder secretes or excretes cholesterol into the bile; and (2) That the gall-bladder reabsorbs a portion of the cholesterol already present in the bile. There is much to suggest that the latter is the correct view.

The strict localization of the deposit to the gall-bladder itself points to this mechanism, which is in accord with the known absorptive property of the gall-bladder in regard to several other substances; and also the occurrence of cholesterol and other lipoids in the epithelium *of the tips of the villi* suggests that an absorption from the lumen is the route involved.

In the experimental field efforts have often been made to demonstrate the absorption of cholesterol from the bile, but these efforts have had little success. Other lipoids, on the contrary, can readily be shown to be absorbed into the gall-bladder mucosa.

Absorption of Other Lipoids.—The demonstration of lipoid absorption by the gall-bladder wall was first given by Aschoff[19] and later repeated by Mentzer.[4] The former introduced olive oil, butter, and sterile milk into the gall-bladders of dogs, the cystic ducts being ligated to prevent expulsion of the inoculum. In each case it was found that after a period of a few days lipoid deposits were present in the epithelial cells, whereas in control animals, in which simple ligature of the duct was performed, no such appearance was seen. Mentzer noted similar findings. Even after as short a period as half an hour the lipoid could be seen in the epithelial cells, and after longer periods it could be followed to the stroma and even to the vascular endothelium. No particles of fat could be observed in the lymph node draining the gall-bladder, and Mentzer therefore concluded that the absorption took place directly into the blood-vessels.

The following experiments are typical of many that have been carried out, using a variety of fatty and lipoid substances.

Experiment 3.—In a cat the gall-bladder was exposed by laparotomy, and the bile removed by a needle inserted at the fundus. In place of the bile about 1 c.c. of oleic acid (insufficient to distend the gall-bladder completely) was injected, the needle puncture being then closed by a fine silk ligature. To prevent expulsion of the lipoid, the common duct was ligated close above its entrance into the duodenum.

[In other experiments the cystic duct instead was ligated, with similar results. A disadvantage of ligating the cystic duct is that a certain amount of damage to the surrounding lymphatic vessels draining the gall-bladder is inevitable; on the other hand, ligation of the common duct introduces the complication of a complete biliary obstruction, with consequent raising of the pressure within the gall-bladder. Control experiments, using both sites for ligation, were therefore always carried out.]

Two days later the animal was killed. The mucosa of the gall-bladder appeared undamaged, but throughout the entire organ it showed numerous milky-white, tiny deposits, scattered throughout the mucosa along the summits of the ridges—an appearance closely similar to that of a typical clinical 'strawberry' gall-bladder.

Microscopically this similarity was confirmed (except, of course, as regards the nature of the lipoid). In sections stained by osmic acid or by Sudan IV, innumerable fatty deposits were visible in the epithelial cells of the mucosa, in exactly the situation most characteristic of cholesterosis—namely, at the bases of the cells

just below their nuclei, and especially in those cells situated towards the apices of the 'villi'. In other sections the lipoid was sometimes found also in the more superficial parts of the cells, and in some the epithelium was completely loaded with fatty particles (*Fig.* 143).

The whole appearance of such sections so strongly resembles that of early cases of cholesterosis that it is difficult to believe that in the latter condition the cholesterol can have reached its site of deposition except by absorption from the lumen into the epithelium of the mucosa. The only respect in which the clinical condition differs from that induced experimentally is in the nature of the lipoid, which in the experiment here cited had the staining reactions of a fatty acid and failed to show anisotropism.

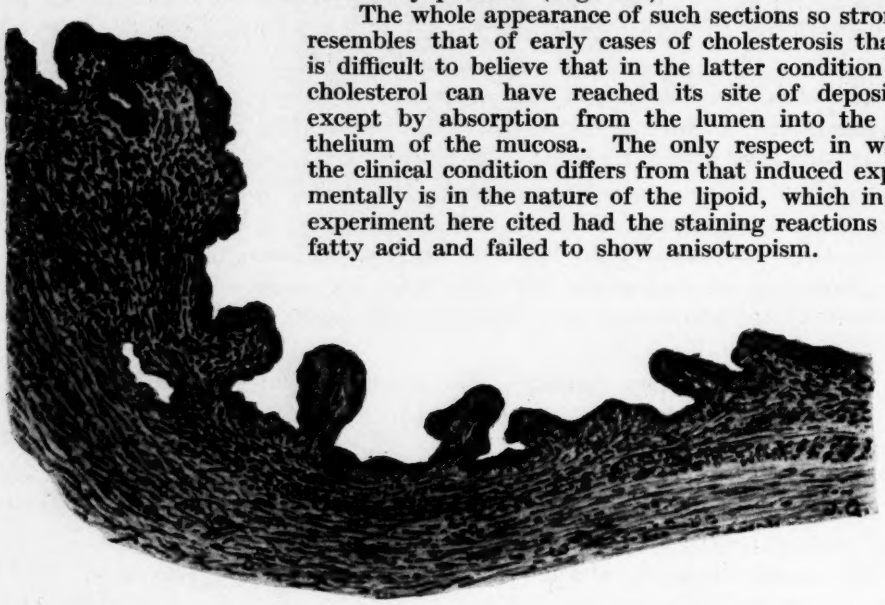


FIG. 143.—Absorption of lipoids by the gall-bladder. Frozen section of the gall-bladder of a cat (stained with Sudan IV). In this experiment the gall-bladder was partly filled with a lipoid (oleic acid), the cystic duct being tied to prevent emptying. This section (taken two days later) shows how a large amount of the lipoid has been absorbed into the cells of the mucous membrane. Olive oil, butter, and several other fatty substances behave similarly. The demonstration of the absorption of cholesterol is more difficult. (× 120.)

In similar experiments the absorption of butter, olive oil, and lecithin has been demonstrated.

Absorption of Cholesterol.—The demonstration of the absorption of cholesterol through the gall-bladder wall has proved difficult. Aschoff in 1906 showed that if cholesterol in olive oil is placed in the gall-bladder, though lipoid appears in the epithelial cells, it does not have the reactions characteristic of cholesterol. Mentzer performed similar experiments, using cholesterol ester, with equally negative results. In addition, cholesterol dissolved in oleic acid was used, and in this case it is stated that absorption took place, though it was not so marked as in the case of other lipoids. Apart from the simple statement, however, Mentzer advances no proof that the absorbed lipoid really was cholesterol, and, unless polariscope examinations are carried out, a fallacy may here creep in.

Experiment 4.—In a cat the bile in the gall-bladder was replaced by 1 c.c. of oleic acid, in which was dissolved 0·2 grm. of pure cholesterol. The common duct was ligated. Two days later the mucosa of the gall-bladder showed evident absorption, and microscopically there was lipoid in many of the epithelial cells. Polariscope examination showed, however, that this lipoid was not anisotropic, and even chemical tests of ethereal extracts of the gall-bladder wall showed but faint traces of cholesterol.

Working from another aspect, Torinoumi[20] has shown how difficult it is to prove that the gall-bladder can absorb cholesterol. Torinoumi, working with dogs, estimated the total cholesterol content of the bile in the gall-bladder before and at the end of a period during which the cystic duct was closed by ligature, and endeavoured to determine whether any reduction of this total took place. Several difficulties attended the performance of these experiments, especially the accidental production of inflammation in the gall-bladder, and the findings were therefore very variable. In those experiments, however, where at the end of the period the gall-bladders appeared healthy, there was a distinct diminution in their cholesterol content, so they do give some evidence of an absorptive function.

It is evident that the proof of the absorption of cholesterol, if it does occur, must be difficult to obtain, for the following reasons :—

1. *The experiments of Torinoumi indicate that absorption is very limited in extent*, possibly depending partly upon a relatively high concentration of cholesterol in the bile as compared with the blood, and slowing down as these concentrations equalize.

2. *The absorption may not be demonstrable by histological methods.* Little is known about the normal method of transport of cholesterol through the body, but it seems probable that the cholesterol is usually bound up with other lipoids in such a manner as to render it unrecognizable by its staining or physical properties. This masking of cholesterol is very evident in absorption through the intestinal mucosa. Thus, in a rabbit which for several weeks had been fed with cholesterol, microscopic sections from several regions of the intestinal tract showed no trace of doubly refractile material, although at the time of death some absorption of cholesterol must undoubtedly have been taking place. It seems possible that the passage of cholesterol through the gall-bladder mucosa may be similarly obscured.

3. In attempts to demonstrate experimentally absorption by the gall-bladder, the physical and chemical state and the environment of the cholesterol used must be of great importance and should correspond as nearly as possible to that present in the normal bile and tissues.

In the course of this investigation numerous experiments have been carried out with the object of giving histological demonstration of cholesterol absorption, but so far they have been entirely unsuccessful. In these experiments, which were carried out on similar lines to *Experiment* 3 above, cholesterol in a great variety of mixtures was used. Crystalline cholesterol, suspended in vaseline, agar-agar, or butter; solutions of cholesterol in oleic acid or olive oil; complex suspensions with lecithin and other lipoids; and extracts of adrenal glands containing much cholesterol: all gave the same results—namely, that absorbed lipoid could be readily demonstrated in the gall-bladder wall, but this lipoid failed to respond to the tests for cholesterol. Although histological proof has been found lacking, it has been possible to demonstrate very clearly the absorption of cholesterol by other methods.

Experiment 5.—A supply of cholesterol, mixed with other lipoids as nearly as possible in the state in which it normally exists in the body, was obtained as follows: The adrenal glands were removed from a number of rabbits which for three months

had been on a diet rich in cholesterol. Frozen sections of these glands showed a large excess of cholesterol, chiefly in the form of esters. The glands were extracted in the cold with chloroform-ether mixture, and a sticky extract was obtained which also showed a high content of cholesterol esters. This extract was emulsified by means of lecithin in a small quantity of cat's bile.

Laparotomy was then performed on two cats, and the same procedure carried out in each. The cystic duct was carefully exposed and freed from the cystic artery, and cut across. With a fine Record needle inserted along the cystic duct the bile in the gall-bladder was withdrawn, and in place of it 1 c.c. of the prepared bile was injected. The cystic duct was then doubly ligatured and the wound closed.

Five days after operation both cats were killed. The gall-bladders, which appeared healthy, free from adhesions, with unimpaired blood-supply and lymph drainage, and with no evidence of leakage, were removed and their total contents carefully collected. Cholesterol estimation was then performed upon these contents, and at the same time upon 1 c.c. of the original injected fluid, all the steps of the three estimations being carried out together to minimize any error.

The results of this experiment are seen in *Table VI*, which shows that during the space of five days more than half of the cholesterol injected had gone from the gall-bladder. This amount far exceeds any possible error of estimation, and as every care was taken to prevent loss during the operative procedure or at autopsy, it seems justifiable to attribute it to actual absorption through the gall-bladder wall.

Table VI.—Absorption of Cholesterol from the Gall-bladder.

	Cat 89	Cat 98
Cholesterol injected into gall-bladder ..	34·425 mgrm.	34·425 mgrm.
Cholesterol present after five days	15·67 mgrm.	13·29 mgrm.
Amount absorbed ..	18·755 mgrm.	21·135 mgrm.

It is concluded, therefore, from these experiments that cholesterol, when in excess and in certain physical mixtures, can be absorbed by the gall-bladder wall, probably in masked form, which prevents its histological demonstration.

DISCUSSION.

It is believed that there are now sufficient data available to justify a tentative opinion as to the etiology of cholesterosis.

The fact that similar infiltrations with cholesterol in various tissues of the body are frequently or usually associated with two demonstrable lesions—namely, an increase in the cholesterol content of the blood and an inflammatory change—necessitates a careful consideration of these factors in particular.

It cannot be doubted, for the reasons already cited, that neither of these factors alone can give rise to cholesterosis, but it is an attractive hypothesis, and one frequently maintained, that they are jointly responsible. Unfortunately this does not completely agree with the available facts, for either hypercholesterolæmia or cholecystitis may be lacking, the former indeed commonly.

In addition, the frequent cases in which hypercholesterolæmia and mild cholecystitis exist, yet fail to give rise to cholesterosis, indicate clearly that other causative factors must be sought for. It seems probable that an indication of the nature of these may best be obtained by a careful consideration of the normal functions of the gall-bladder in relation to cholesterol.

From the experiments described above it seems clear that the gall-bladder can absorb cholesterol from the bile. This does not necessarily mean that such an action goes on to any appreciable extent in the normal gall-bladder (the experiments of Torinoumi indicate that such action must at least be very limited in degree), and it is likely that an essential feature in determining the absorption of cholesterol is the concentration of this substance in the bile, absorption taking place only when it is in excess. Moreover, it is clear that the cholesterol, when absorbed into the healthy mucosa, yet remains indistinguishable under the microscope, its characteristic optical properties being masked in some way not fully understood.

This being the case, it becomes evident that deposit of visible cholesterol in the gall-bladder (cholesterosis) postulates essentially two processes: (1) Absorption of cholesterol into the mucosa, depending probably upon an increase in the cholesterol content of the bile; and (2) Some change which unmasks this absorbed but invisible cholesterol, and which furthermore prevents or delays its transport and leads to its accumulation in the mucous membrane.

At this point we may see how it is possible to link up this hypothesis with clinical observations.

1. An increased cholesterol content of the bile was found clinically in all the cases in which the estimation was carried out. In 17 of the total cases, moreover, it had progressed to the formation of stones. This cholesterol increase *in the bile* is regarded as a primary factor of importance; the blood-cholesterol may or may not be increased. In the experimental production of cholesterosis (as in *Experiment* 2) an increase of blood-cholesterol is a necessary intermediate step in raising the bile-cholesterol, but there is much evidence that in man the latter alone may be increased.

2. In the second essential process we can see how the factor of cholecystitis may be linked up with the pathogenesis of cholesterosis, for it is believed that the part played by the inflammation consists simply in interfering with this absorptive process, so that the absorbed cholesterol is rendered visible and at the same time is accumulated in large quantity in the gall-bladder wall.

The exact rôle of the inflammation is open to speculation. Chiray and Pavel[8] have suggested that it acts by actual blockage of the lymphatic drainage of the gall-bladder; but against the acceptance of this hypothesis are two facts, namely: (1) There is no proof that absorption usually occurs by the lymphatic path; (2) There is no histological evidence of lymphatic obstruction and no œdema or fibrosis of the gall-bladder wall.

A more feasible hypothesis would appear to be that the inflammatory process occurs in virtue of some chemical action directly upon the absorbed cholesterol which would at the same time render the cholesterol visible, interfere with its normal transport, and lead to the characteristic endothelial

response. Such a hypothesis has the additional advantage that it is also applicable to those cases in which no inflammatory element is recognizable. In these cases one may presume some non-inflammatory change in the nature of the absorbed cholesterol, which prevents its transport away from the gall-bladder.

SUMMARY.

1. Cholesterosis of the gall-bladder, which includes the so-called 'strawberry' change and also cholesterol polyposis, consists essentially of an infiltration of the epithelium and the stroma of the mucous membrane with lipoids, and especially with cholesterol. In the stroma a characteristic feature is the presence of large 'foamy' cells of endothelial origin.

2. Cholesterosis is of frequent occurrence. It is a condition chiefly of middle life, and the incidence bears no relation to sex or social status.

3. It is usually associated with cholecystitis, especially of mild degree. Gall-stones are frequently present, especially 'pure' cholesterol stones.

4. Cholecystography indicates that in uncomplicated cases two functions of the gall-bladder (concentration of the bile, and the emptying in response to fats) are not affected.

5. The cholesterol content of the blood is raised in some cases, but is often normal.

6. Symptoms, even in uncomplicated cases, are extremely varied, and the diagnosis is correspondingly difficult.

7. Treatment by cholecystectomy is the most rational procedure, and appears to yield satisfactory results.

8. Experiments are described which indicate: (*a*) That cholesterosis may be most readily brought about in the rabbit by the association of a prolonged state of hypercholesterolæmia with a mild chronic bacterial cholecystitis; (*b*) That cholesterosis does not result simply from the deposit of an excess of cholesterol from the blood, but is intimately linked up with the function of the gall-bladder in regard to cholesterol.

9. In an experimental investigation of the absorbing function of the gall-bladder it has been shown: (*a*) That absorption of several other lipoids from the bile is easily demonstrable; (*b*) That cholesterol is absorbed in unrecognizable form, its presence in the mucosa being obscured, probably by admixture with other lipoids. Absorption of cholesterol from the bile probably only occurs when it is present in excess.

10. The conclusion is drawn that cholesterosis results from two essential primary changes: (*a*) An increase in the cholesterol content of the bile, which leads to the absorption of cholesterol into the mucous membrane of the gall-bladder: this increase depends probably upon several factors which are incompletely understood. In some cases it is associated with a similar increase of the blood-cholesterol. (*b*) A change in the physical and chemical state of the absorbed but invisible cholesterol, which renders it optically active and recognizable, and which by preventing its transport leads to its accumulation in the gall-bladder wall. This change is most frequently due to an inflammation of the gall-bladder.

In conclusion, I would acknowledge my great indebtedness to Professor D. P. D. Wilkie for the facilities of his Department and for personal encouragement and help, to Professor Lorrain Smith for kindly advice and criticism, and to the technical staff of the Department of Surgery for their skilled assistance.

Part of the research was carried out during the tenure of a part-time grant from the Medical Research Council, and the expenses were defrayed by grants from the Earl of Moray Endowment for Research.

REFERENCES.

[1] Virchow, R., *Virchow's Arch.*, 1857, ii, 574.
[2] Moynihan, B. G. A., *Ann. of Surg.*, 1909, l, 1265.
[3] MacCarty, W. C., *Ibid.*, 1910, li, 651.
[4] Mentzer, S. H., *Amer. Jour. of Pathol.*, 1925, i, 383; *Surg. Gynecol. and Obst.*, 1926, xlii, 782.
[5] Boyd, W., *Brit. Jour. Surg.*, 1922–3, x, 337.
[6] Bergeret, A., and Dumont, J., *Presse méd.*, 1927, xxxv, 177.
[7] Gosset, A., Loewy, G., and Magrou, J., *Bull. et Mém. Soc. de Chir. de Paris*, 1921, xlvii, 1391.
[8] Chiray, M., and Pavel, I., *La Vésicule biliaire*, 1927. Paris: Masson et Cie.
[9] Lecène, P., and Moulonguet, P., *Presse méd.*, 1926, Jan., 49.
[10] Stewart, M. J., *Jour. Pathol. and Bacteriol.*, 1915, xix, 305; *Brit. Med. Jour.*, 1924, ii, 893.
[11] Naunyn, B., *Die Entstehung u. d. Aufbau d. Gallensteine*, 1923, Jena.
[12] Dyke, S. C., *Jour. Pathol. and Bacteriol.*, 1928, xxxi, 173.
[13] Major, R. H., *Johns Hopkins Hosp. Bull.*, 1924, xxxv, 27.
[14] Anitschkow, N., *Münch. med. Woch.*, 1913, xlvi, 2555.
[15] Pick, L., *Berl. klin. Woch.*, 1908, xxxvii, 1679.
[16] Daniel, C., and Babès, A., *Presse méd.*, 1923, Dec., 1073.
[17] Blaisdell, F. E., and Chandler, L. R., *Amer. Jour. Med. Sci.*, 1927, clxxiv, 492.
[18] Wilkie, A. L., *Brit. Jour. Surg.*, 1928, xv, 450.
[19] Aschoff, L., *Münch. med. Woch.*, 1906, xxxviii, 1847.
[20] Torinoumi, K., *Ziegler's Beitr.*, 1924, lxxii, 456.

SPLENECTOMY FOR EGYPTIAN SPLENOMEGALY: AN ACCOUNT BASED ON A RECORD OF 390 CASES.*

By H. E. S. STIVEN,

PRINCIPAL MEDICAL OFFICER TO THE EGYPTIAN GOVERNMENT HOSPITAL, PORT SAID.

Splenomegaly amongst the Egyptian fellaheen is one of the most disabling diseases to which they are prone. There is no doubt in my mind that this enlarged spleen is due to heavy infections of bilharzia of the rectal or *Bilharzia mansoni* types. Proof of this statement is quite another matter, but I am confident it will be forthcoming, and my only regret is that I have no opportunity for field work in this connection, but presently I feel assured the Bilharzia Committee will discover the clue.

I have been performing splenectomy now for the last nine years, and have removed a total of 390 spleens, and I must acknowledge my debt to Owen Richards, at one time surgeon at Kasr el Aini Hospital, who did the preliminary work on this operation, and it is largely through his experiences that I am now accorded a certain measure of success. But it is astonishing how difficult it is to impress the young surgeon with the difficulties of the operation and the precautions that have to be taken, with the result that they get a hundred per cent mortality in their first few cases, and naturally become frightened, and persuaded that the operation is not worth the risk.

It is readily accorded that this procedure is extremely dangerous: not only is the operation itself a dangerous one, needing a gentle hand, a quick eye, and coolness in an emergency, but the material to work on is of the unhealthiest nature. For this reason it cannot be too forcibly impressed upon surgeons wishing to perform this service that the patient must be very carefully prepared before being subjected to the risk of the operation.

I find surgeons examining the stools and urine of these anæmic patients, and if no bilharzia ova or ankylostoma eggs are found, they proceed to operate at once, with fatal results. Whether eggs are found or not, it must be presumed that the patient is full of the common Egyptian parasites, and carbon tetrachloride and a full course of tartar emetic and a course of antisyphilitic treatment must be given and the patient built up by nourishing food. This entails a stay in hospital for at least one month to six weeks. After all this preparation my immediate mortality (by immediate I mean those patients who do not leave hospital alive) is 13 per cent. On following-up patients I find this figure increased to 19 per cent by those who die after getting back to the villages—that is, within two or three months.

I do not choose my cases, but operate on all that come to me if they can

---

* A Paper read at the International Congress of Tropical Medicine and Hygiene, Cairo, 1928.

possibly stand the operation, even patients with ascites, which is an unfavourable factor. By choosing cases this mortality can be reduced to 5 or 6 per cent, but 20 per cent will compare favourably with the 40 to 50 per cent mortality of the pioneers in surgery of cerebral tumours, and doubtless we shall reduce our mortality rate as they have reduced theirs in the light of experience.

The difficulty in a country like Egypt is the following-up of patients, and without a good follow-up system a surgeon is very apt to get wrong ideas of his work and statistical results. I have endeavoured by a system of questionnaires sent each year through the police to the omdah or headman of the village to get replies from my cases for three years in succession. The questionnaire is simple: Is the patient still alive? Has he increased in weight? Does he feel better than before the operation? This paper is returned to me:

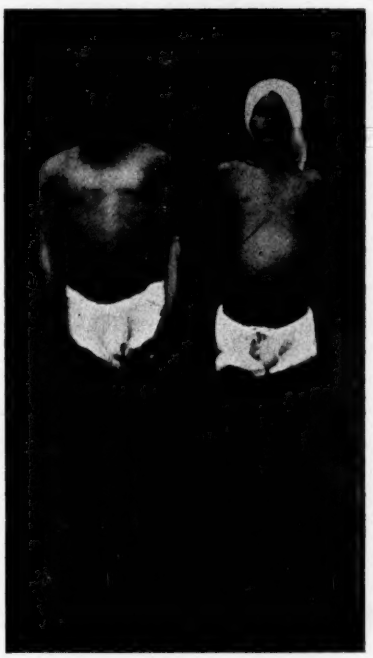


FIG. 144.—A typical case of splenomegaly before operation, with another case one year after splenectomy.

FIG. 145.—Photograph of a patient thirteen months after operation.

I find no trace of 6 per cent of my cases, indifferent health in 5·5 per cent, death in 19·5 per cent, good reports in 69 per cent. A good report of 69 per cent is my justification for allowing the patient to undergo the grave risk of the operative procedure, because it must be understood that without operation their expectation of life is extremely limited. *Figs.* 144, 145 show the good results that can be obtained.

The sexes are probably equally involved, but naturally in Egypt the women do not come forward so readily for treatment as the men, their economic value being so much less. Neither is the accommodation in our hospital sufficient for their needs. My cases show 80 per cent men. The ages vary from 8 to 45, the majority being between 15 and 25.

The geographical distribution is shown on the map (*Fig.* 146), but more work has to be done on this question. I am inclined to think that villages

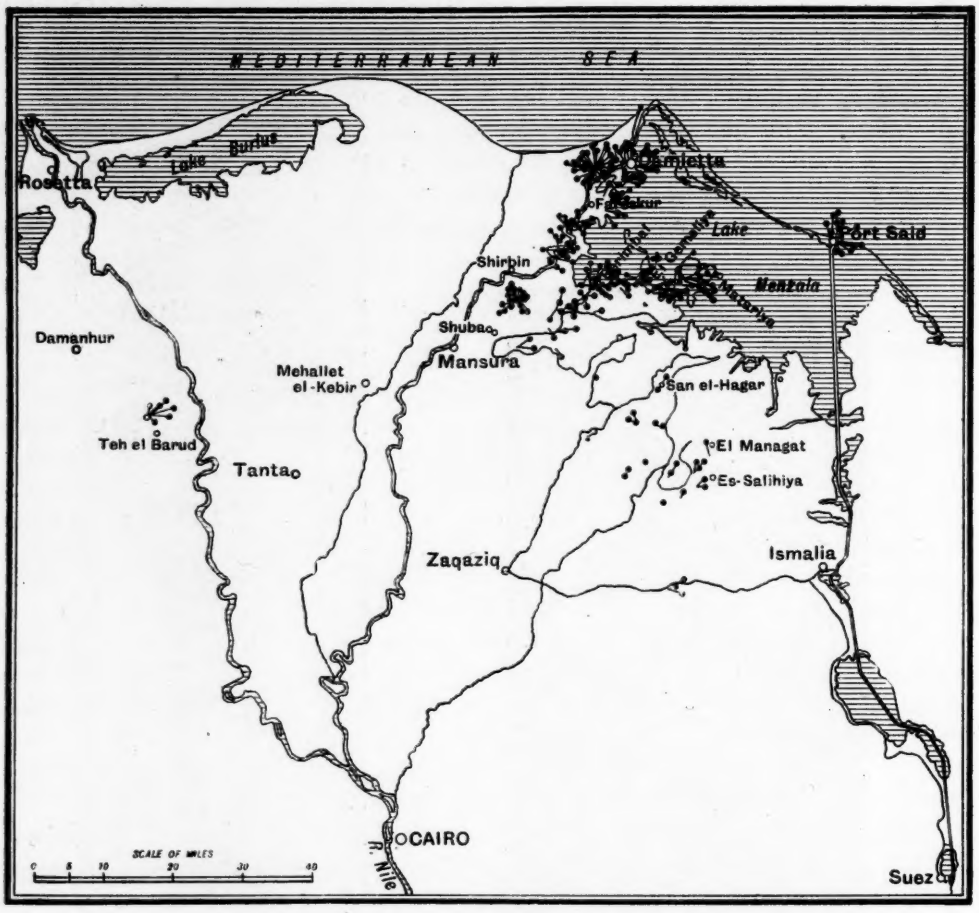


FIG. 146.—Map illustrating the geographical distribution of cases operated upon.

at the end of canals are more heavily infected than elsewhere; but proper surveys, with the help of the Irrigation Department and the Entomological Department as to the distribution of the snails, will demonstrate this more clearly.

Blood-counts on patients will show white count 6500 and red count one or two millions. A differential count will give: polymorphs 66 per cent, lymphocytes 20 per cent, large mononuclears 6 per cent, transitionals 6 per cent, eosinophils 1 per cent, mast cells 1 per cent. The fæces show *Bilharzia*

mansoni, and the urine may be infected also with *Bilh. hæmatobia*, and in addition the fæces may show amœbæ, trichomonas, ascaris, and all the flora and fauna of the country.

The weight of the spleens removed averages 1½ kilo., and varies between 800 grm. and 7 kilo.

PREPARATION OF THE PATIENT.

When a patient is admitted for splenomegaly with a view to operation I give as a matter of routine a dose of carbon tetrachloride varying from 2 to 4 grm. according to age and weight; a full course of tartar emetic intravenous injections 0·12 grm. every two days for twelve injections; and a course of injections of '606' or corresponding drug. I give a mixture of rhubarb and soda for the first week (this is a splendid cleaner of the intestinal tract), and subsequently a mixture of iron and arsenic and a full nourishing diet including 'fool nabet', that is, beans which have been allowed to germinate forty-eight hours in water. These are cooked quickly—four or five minutes only—in a good soup. They have a remarkable effect on the pellagrous cases and others suffering from a vitamin-deficient diet. This course of treatment lasts from five to six weeks, when the patient is greatly improved in general health and can stand the shock of operation.

THE OPERATION.

The evening before operation the patient is purged and given an injection of pneumococcic vaccine. This is essential, as, failing this, the patient will almost certainly die from pneumonia after the operation. In fact, about 5 per cent have pneumonia in spite of the vaccine, but with another dose of vaccine they generally pull through. A dose of morphine and atropine is given half an hour before the operation.

The patient sits on the operating table and places his hands over his ears and bends down his head. This opens up the spinal vertebræ, and with a long fine needle on a 2-c.c. syringe, I tap the spinal fluid through the eleventh dorsal space, trying not to let any of the fluid escape and drawing it into my syringe. I then inject about 1·8 c.c. of the stovaine solution, using stovaine 0·04 grm., sodium chloride 0·0011 grm. per c.c.

The patient lies down promptly on his back, and I do not practice raising the legs or lowering the head, as I find there is a smaller percentage of headache after the operation by omitting these procedures. The anæsthesia is good and sufficient for the whole operation; only very occasionally is a little chloroform needed in addition.

Standing on the right hand side of the patient, I make an incision varying in length with the size of the spleen. This incision starts at the costal margin and runs parallel to the mid-line, dividing the left rectus muscle into two equal parts. I incise the skin and rectal sheath, and make a small incision through the intermuscular septum and separate the muscle with a sweeping action up and down with my finger. The peritoneum is then incised. The exciting part of the operation now follows, as the whole hand

is inserted to find out what adhesions are present between the spleen capsule and the parietes, as the difficulties of the operation depend on the adhesions, and it is impossible to foretell the nature and extent of these obstructions.

In a favourable case, with no adhesions, or only slight easily detached ones, the whole spleen can now be delivered out of the abdominal wound. A dozen big clamps are ready on the instrument table, and I clamp the pedicles of the splenic artery and vein all together with three or four clamps placed in juxtaposition, cutting off the spleen between the third and fourth clamp. The pedicle is then transfixed with a long pedicle needle threaded with a black and white linen thread—a point I learnt from T. de Martel in Paris. Linen is better than silk, and ties in a knot that does not slip. Black and white threads enable one to know at a glance which is which and whether the ligatures are interlocked or not. This first ligature is put in the space revealed by releasing No. 2 clamp, and as it is tied I release No. 1 clamp and allow it to be tied tight. Another ligature is then tied round the whole pedicle in the position of No. 1 clamp, and then a third is tied in place of No. 3 clamp. Various modifications of this procedure have to be undertaken according to circumstances. Very often there is a large vessel running from the greater curvature of the stomach to the hilum of the spleen. I pick up the lesser omentum and tie it off, and thus expose the true pedicle of the spleen.

Frequently there is a very strong adhesion from the splenic flexure of the large intestine to the spleen, and sometimes there are strong adhesions between the spleen and the under-surface of the diaphragm. These have to be broken by the fingers, and after the removal of the spleen, my assistant putting in a wide retractor, I have to pick up these bleeding points with a long bullet-nosed clamp and tie them off. Care is taken to stop all bleeding and clean out the cavity before shutting the abdomen.

The peritoneum is sewn with a continuous chromic catgut No. 0 or 1, and then, with a long Reverdin's needle, three or four supporting silkworm-gut sutures are put in through the skin and the rectus muscle. I then join the tendinous septum of the rectus with a catgut stitch, and subsequently sew up the external sheath of rectus with continuous chromic catgut No. 1 or 2. The skin is closed with Michel's clips and the silkworm gut tied, leaving the ends long, which are again tied over a sausage-shaped roll of dressing to act as a splint.

I have had no cases of paralysis of the inner portion of the rectus muscle in spite of the nerve-supply being divided. I think that breaking the muscle fibres apart with the fingers, instead of cutting, probably pulls the nerve-fibrils out and leaves them to spread quickly across the wound during repair; also, I take it, muscle fibres being contiguous go into contraction with the outer half of the muscle out of sympathy. I am not in favour therefore of the incision which has been suggested for this operation—namely, one starting at the tip of the eleventh rib and running transversely across towards the umbilicus and dividing the rectus muscles. I find no particular advantage in the exposure of the field of operation, and there is peculiar difficulty in sewing up the divided rectus.

After operation patients are kept without anything to drink for twelve hours, my experience being that, given the chance, they will fill their

stomachs with water, and may start hæmorrhage from some unsuspected vessel. As a rule no rectal saline or intravenous blood transfusion is given, as I find that, although the patient may have lost a large amount of blood, he soon makes it up again.

Diet from the beginning is strictly fever diet, and only after five days is a purge given and a nourishing diet commenced. As a rule, patients go out after fifteen days, weak and happy, with a bottle of iron and arsenic, relieved of the intolerable weight in their abdomens, and in two or three months are really fit for work.

CONCLUSIONS.

1. Splenectomy is a dangerous operation requiring especially careful preparation of the patient.

2. The improvement in general health only to be obtained by its means justifies the risk.

THE CAUSATION OF MULTIPLE EXOSTOSES.

By J. B. HUME, London,

This condition has been variously described as multiple exostoses, multiple endosteomata, and diaphysial aclasis. All these terms describe to some extent the pathological process involved, for projecting exostoses are present, the cancellous bone is of irregular and abnormal growth, and the ends of the diaphysis are unmodelled. The occurrence of these multiple irregular outgrowths at the ends of the long bones, associated with deficient growth in length and irregularity of contour, has long given rise to dispute among those interested in the problems of bone growth and bone pathology. The problem of modelling is itself ages old, for John Hunter,[1] in his lectures on the "Principles of Surgery", wrote: "Absorption must necessarily go on to keep the bone in its proper shape, hence I call this the modelling process."

Fig. 147.—The bones of the left forearm of a boy, age 14. Note the tilting of the lower radial epiphysis, the absence of the lower ulnar epiphysis, and the inequality in length of the bones.

Many cases of this condition have been described, and it is well known to all surgeons and pathologists, so that only a brief résumé of its salient features as affecting the present problem will be given.

No definite information is available as to the exact date of appearance of multiple exostoses. They are not found in embryos or fœtuses, but cases are recorded at the age of two. When the lower end of the ulna is the site of an exostosis the normal ossification of its epiphysis is interfered with, indicating that the process is well established before the age of three (*Fig.* 147). Either sex may be affected. The subjects are of less than the normal stature, owing to the deficient growth of their long bones. Certain bones are never affected; the carpus and tarsus, vertebræ, sternum, and skull are always free from any abnormality, and also the epiphyses of the long bones. It is thus clear that bones developed purely within cartilage or membrane escape, and that only those in which there is additional membrane bone laid down subperiosteally over endochondral bone are affected. The condition is often hereditary, though a history of this is not always obtained. An association with rickets has not been established, but there is a definite association with the occurrence of multiple enchondromata.

Types of Multiple Exostoses. — A number of different forms of multiple exostoses may be recognized, frequently occurring in the same case. The most common are those which occur at the extremities of the long bones, and appear as globular or cauliflower-shaped projections from the cylindrical mass of unmodelled bone (*Fig.* 148). The projections may also take the form of elongated spikes, in which case they are always directed away from the epiphysis, and are nearly always broader at the base than at the apex (*Fig.* 149). In this respect they differ markedly from the single pedunculated exostosis.

In addition to those originating from the region of active growth, we find exostoses occurring near the secondary centres of ossification, such as the gluteal ridge of the femur or the vertebral border of the scapula. These are usually cauliflower-shaped. A few seem to have no connection with any ossification process, and appear as small isolated projections from the centre of the shaft of a long bone, frequently at points of tendon insertion or of muscle origin. These are usually pedunculated. It should be noted that these single exostoses occurring from points of tendon insertion or of muscle origin are the only ones which project from a normally shaped area of bone.

FIG. 148.—A lateral view of the knee-joint of a youth, age 17. Note the large cauliflower-shaped exostosis on the femur, the globular one on the tibia, and the translucent appearance of the upper end of the tibia.

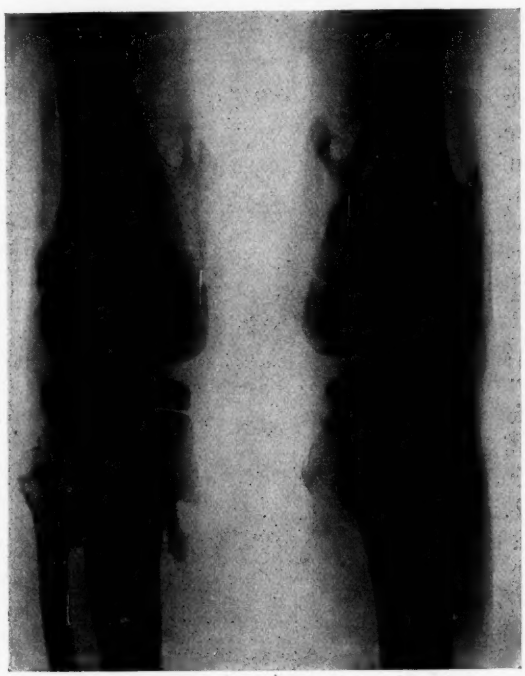


FIG. 149.—The knee-joints of a girl, age 9, showing spiculated exostoses. The alteration of arrangement in the cancellous bone of the affected area is well shown.

Structure of Exostoses.—It is instructive to recall the mode of growth of the common single pedunculated exostosis, unassociated with any other skeletal abnormality. It is considered likely that a portion of the periphery of the growth disc has become displaced on to the

surface of the shaft, possibly by the pull of the periosteum attached to it. It then continues to grow, its main growth taking place at a right angle to the growth disc. The resultant of the forces in the two directions of growth finally brings the exostosis into a line directed away from the growth disc and at an angle to the shaft of about 30°. It grows by means of a cap of rapidly dividing cartilage which covers its enlarged extremity, and is identical in appearance with a growth disc (*Fig.* 150). The stalk or shaft of the exostosis is always narrower than the cap of cartilage which gave rise to it. Ossification of this cap of cartilage occurs about the age of 20.

FIG. 150.—A pedunculated exostosis growing from a bone of normal contour by means of a cap of cartilage. (Diagrammatic.)

Multiple exostoses are also covered by a thin layer of cartilage until the age of 20, when this layer is replaced by an egg-shell covering of compact bone. The cartilage-covered area extends from the diaphysial growth disc to the point at which the normal contour of the shaft begins, which is always marked by the presence of an out-turned spur or lip (*Fig.* 151). Sections of this surface layer show that the cartilage cells are behaving in the same way as the cells of the diaphysial disc—that is to say, they are dividing and adding more and more cells on their deep surface. Some of these cells are being replaced by compact bone, which forms an incomplete layer beneath the cartilage. Many gaps are present, and these frequently contain large multinuclear cells. The appearance of the type of growth occurring from the surface cartilage closely resembles that occurring from the diaphysial disc in rickets, though this fact should not be construed into an association between the two conditions.

The cancellous bone underlying the surface cartilage is excessively spongy and fragile, the spiculæ are small and irregular, and the interstices are large and filled with myxomatous tissue. The irregularity of the trabeculæ in the cancellous bone is well shown in radiographs, and may be compared with the characteristic appearance of normal cancellous bone, which has been beautifully illustrated by Murk Jansen.[2]

Bone Modelling.—The process of bone modelling has long been an embryological mystery. The bone once formed increases in length by apposition from the growth disc, and in diameter by the apposition of subperiosteal bone. As new bone cells are formed

FIG. 151.—The right hand of a boy, age 14. Note the out-turned spurs marking the termination of the compact bone at the lower ends of the radius and ulna. Several of the phalanges are involved.

from the growth disc, the broad end adjacent to the epiphysis is displaced towards the centre of the shaft. It has always been assumed that the portion of bone which originated from the periphery of the disc must be removed in order to maintain the contour of the shaft. This process is referred to by Jansen[3] as 'tubulation', and is explained as being brought about by the active remodelling of osteoclastic cells (*Fig.* 152). This whole conception is contrary to the new evidence obtained from tissue culture. Miss Fell[4] has shown that the cartilage cells from a growth area arrange themselves in a definite manner which determines the shape of the bone. It is not that certain peripheral cells are removed in order to produce the normal contour of a long bone, but that the process of cell growth is so perfectly balanced as to bring this about. The two processes of the formation of perichondral and endochondral bone from the subperiosteal tissues and the growth areas respectively go on hand in hand. As there is no evidence of the occurrence of multiple exostoses in the embryo or the fœtus, and as the condition is not usually noticed until the third year or later, it is fair to assume that it is not due to any congenital defect in the system of bone modelling.

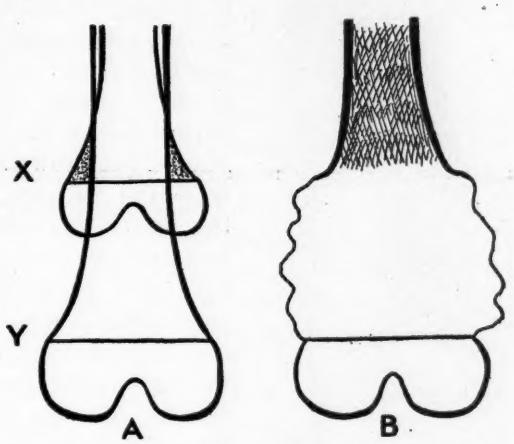


FIG. 152.—A, Illustrating Jansen's tubulation theory. The stippled area represents the portion of bone to be removed during the process of increase in length from X to Y. B, A bone equal in length to Y, showing how the out-turned spurs indicate the position of the growth disc immediately before the process of irregular growth commenced.

Conclusions.—Murk Jansen,[3] in his essay on the " Dissociation of Bone Growth ", ascribes the causation of multiple exostoses to a dissociation of resorption and tubulation, and mentions no connection between the surface cartilage and the growth of the exostosis. If the defect were merely the failure of the osteoclastic remodelling, a normal formation of bone in respect of length and structure would be found, with possible surface irregularity due to defect in the laying down of surface compact bone which goes hand in hand with absorption. The normal length, however, is not found, and irregularities occur both on the surface and in the medullary cavity. Moreover, up to the time of cessation of bone growth, growing cartilage is found on the surface of the exostosis.

The origin of this surface cartilage would appear to be the key to the situation. It is actively growing, giving rise to osteoid tissue and finally, by replacement, bone. Virchow first drew attention to the presence of growing cartilage on the surface of the exostoses, and though his explanation of its derivation is discarded, probably correctly, by Jansen,[3] yet the facts are basic ones and lend themselves to an alternative theory.

The exostoses are most numerous and best marked at the areas where normally the greatest growth of an individual bone takes place, and are most

frequently found at the upper end of the tibia and the lower end of the femur. This association with the areas of growth would suggest the derivation of the cartilage from which the exostoses increase in size to be from the growth disc, the edges of which have become pulled upwards over the surface of the metaphysis. This is analogous to the growth of the single pedunculated exostosis from a cap of cartilage, which is a completely isolated portion of the growth disc. It is a matter for speculation how this spreading outwards and upwards of the edges of the growth disc is brought about. The disc is slightly concave, and the periosteum of the shaft is attached to its circumference. Should the growth of the periosteum and the periosteal bone fail to keep pace with the increase in length of the shaft, the peripheral growing cells become displaced, and in their new situation produce an irregular growth of bone. It is not, however, sufficient to ascribe the condition to a peripheral change alone, as imperfect and irregular growth is taking place from the growth disc as well as from the surface cartilage, and the type of spongy bone laid down by the two sets of cells is identical. The alteration of the normal structure and arrangement of the cancellous bone is very apparent in radiograms of the condition, and normal cancellous bone does not appear until the normally modelled shaft is reached. Growth does not even take place at an equal rate all over the disc, which is proved by the tilting of the epiphysis (*see Fig.* 147). In a bone such as the femur, for example, two points on opposite sides of the shaft would always have, in normal growth, the same relative distance from the growth disc. In multiple exostoses the spurs marking the commencement of modelling are rarely at the same level, indicating that the process commenced first on one side of the disc rather than over the disc as a whole.

FIG. 153.—The hand and forearm of a girl, age 14, showing multiple exostoses associated with multiple enchondromata.

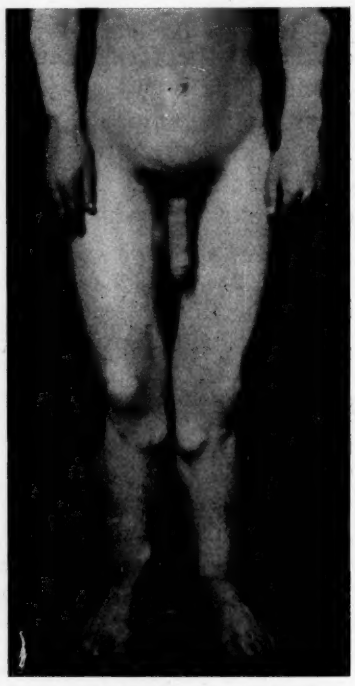


FIG. 154.—A youth, age 17, with multiple exostoses. Note the general deformity of the limbs and the precocious development of the genitalia.

In this connection one point must be considered—the association with multiple enchondromata, which probably originate from a failure of ossification in the centre of the disc (*Fig.* 153). In addition to the occurrence of enchondromata, which are often regarded as innocent new growths rather than as growth defect, there is the rare appearance of an osteogenic sarcoma

in the exostoses. The theory of a neoplastic basis for the whole condition must, however, be rejected, because the growth of the exostoses ceases at 20 or 25. It also affects many bones and is to a large extent hereditary. The probability is that there is some defect in the internal secretions controlling bone growth, such as the pituitary or genital gland secretions. Jansen mentions that some of the cases show gigantism (*Fig.* 154). It is well known how the processes of bone growth can be interfered with by alterations in diet, and in the stimulus provided by the internal secretions.

It is clear that such a complicated condition cannot be produced by a mere failure in tubulation, or by vascular disturbance, but that, as Keith[5] originally suspected, the cause must lie in the abnormal behaviour of the cells of the growth disc, and the consequent failure of the subperiosteal bone formation to keep pace with it. An abnormal stimulus affecting the centre of the disc alone, and interfering with the process of ossification, would produce an enchondroma; one affecting a localized portion of the periphery, a single pedunculated exostosis; while a more general stimulus affecting the growth discs of all the long bones would produce multiple exostoses.

I am greatly indebted to Sir Arthur Keith for his criticism and advice, to Dr. Kingston Barton for some helpful suggestions, and to Dr. Thurstan Holland for a remarkable series of radiographs, some of which have been used to illustrate this paper.

REFERENCES.

[1] HUNTER, JOHN, *Principles of Surgery*, Palmer's Edition, i, 254.
[2] JANSEN, *Bone Formation.*
[3] JANSEN, "Dissociation of Bone Growth", *Robert Jones Birthday Volume*, 1928. London: Humphrey Milford.
[4] FELL, *Arch. f. exper. Zellforsch.*, 1928, vii.
[5] KEITH, "Studies on the Anatomical Changes which Accompany Certain Growth Disorders of the Human Body", *Jour. of Anat.*, liv, Parts 2 and 3.

ENDOTHELIOMA OF THE NASOPHARYNX: AN INFILTRATING TUMOUR AT THE BASE OF THE SKULL.

By A. J. GARDHAM,

ASSISTANT DIRECTOR OF THE SURGICAL UNIT, UNIVERSITY COLLEGE HOSPITAL, LONDON.

The tumours which I refer to under the name of 'endothelioma of the nasopharynx' have long been recognized, and were shortly described in this country as long ago as 1911, by Mr. Wilfred Trotter,[1] in a lecture given before the Medical Society of London. That they have not received more attention is due partly to their actual rarity, partly to the fact that they still frequently pass unrecognized, and partly also to their almost unique unfavourability for surgical removal. The recent introduction into general use of radium treatment by means of 'seedling' tubes may, I think, render their early diagnosis a matter of practical as well as theoretical interest, as there is some evidence that this type of growth is susceptible to the influence of radium, and if the difficulties of its accurate application can be overcome, I believe there is ground for hope in this method of treatment. To carry out treatment by this means it is essential that the surgeon should have an accurate knowledge of the method and direction of spread of the tumour.

The disease is one which can occur within wide limits as regards age, and in either sex. Of the nine cases which I propose to use in order to illustrate this paper, three were women and six men. The ages of two of the men were 29 and 31 respectively; of the rest of the patients, four were between 50 and 60 years of age, and three between 40 and 50. The tumour has been regarded as one peculiarly apt to affect the young adult male, but my series is too small to permit of any conclusions, and all that can be said on the subject is that the tumour may occur in young adults, but may equally well be met with in people of more advanced years.

The clinical manifestations of these tumours are so varied that it is no easy matter to classify them, or to state the clinical evidences which justify the inclusion of any particular tumour in the group. Some cases show a quite unmistakable combination of symptoms, while the claim of other cases to belong to the group rests on the occurrence of only one or two of the clinical signs, combined possibly with histological evidence.

I shall attempt to describe these growths under seven principal headings: (*I*) *The clinical appearance and direction of spread of the primary growth;* (*II*) *The structures invaded by the primary growth;* (*III*) *The clinical characteristics of the glands invaded by the growth;* (*IV*) *Remote metastases;* (*V*) *The early signs and symptoms of the growth;* (*VI*) *The microscopic appearances of the growth;* (*VII*) *Possible lines of treatment.* To illustrate these points I propose to quote the case records of several patients who have come under my notice in the last few years.

I. THE CLINICAL APPEARANCE AND DIRECTION OF SPREAD OF THE PRIMARY GROWTH.

The primary site of the tumour is to a certain extent obscure, and must, in the present state of our knowledge, be deduced from the early symptoms. Reference to the table of early symptoms which appears in the later part of this article suggests that the tumour very soon comes into relationship with the Eustachian tube, the wall of the pharynx itself, and the 5th nerve. This places the site of origin of the tumour fairly accurately, but whether the growth originates actually in the wall of the nasopharynx is not easy to decide. I am not aware that any case has been recorded in which the symptoms of one of these tumours were present without a palpable mass in the lumen of the nasopharynx, but the syndrome is hardly sufficiently well recognized for such an eventuality to attract attention. From the clinical characteristics of the growth it may be said with certainty that it is not an epithelioma arising in the mucous membrane of the pharynx. The term 'endothelioma of the nasopharynx' is sanctioned by usage and is sufficiently accurate, provided that one remembers that the symptoms are usually those of an infiltrating tumour at the base of the skull rather than those of a growth in the lumen of the nasopharynx.

When seen in the nasopharynx the tumour is commonly small and sessile and lies in the lateral wall of the pharynx in the region of the opening of the Eustachian tube. It is pink in colour, firm to the touch, and may be only appreciable to the examining finger as a hardening in the wall of the pharynx. Ulceration is either absent or slight in the early stages, and a study of cases in more advanced stages reveals a curious characteristic of the growth—that is, its tendency to spread widely beneath the mucous membrane of the mouth without causing ulceration. The tumour reaches the mouth, I think, by extension down the levator palati muscle. In any case it appears at the junction of the hard and soft palate on one side, and from this position extends widely in the submucous tissue, producing a very pronounced thickening in the alveolus, sometimes as far forward as the incisor region. The older parts of the tumour may ulcerate, but submucous extension goes far in advance of the ulceration.

Any tumour which spreads in the mouth in this manner is under suspicion of a nasopharyngeal origin, and if the tumour has appeared at the junction of the hard and soft palates and has been preceded by neuralgic pain in the distribution of the trigeminal nerve, the suspicion becomes practically a certainty. In two of the cases which I shall presently quote (*Cases* 4 and 5), persistent pain of trigeminal distribution preceded the appearance of the tumour in the mouth by several months, and I have no doubt that the pain in these two cases was due to the unsuspected presence of a nasopharyngeal growth. The tendency to submucous spread without involvement of the mucous membrane can be seen sometimes in the microscopic sections (*see Fig.* 157).

In exactly the same way as the tumour burrows beneath the mucous membrane of the mouth it will burrow in the deeper planes. This leads to infiltration of muscles, to a wide involvement of the structures at the base

of the skull, and may ultimately show as an obvious extension on the outer surface of the skull. Two of the cases which I have personally observed showed this sign. A photograph of *Case* 2 is reproduced in *Fig.* 155. The filling up of the temporal fossa on the side of the tumour shows in marked contrast to the hollow on the opposite side in a wasted patient, and produces an obvious alteration of contour of the face.

FIG. 155.—*Case* 2. Photograph of the patient J.M., showing the filling up of the temporal fossa on the side of the tumour.

There is no tendency to the formation of a mass of growth in the pharynx at the site of origin of the tumour; in fact it is the exception to see a mass of growth large enough to produce any marked nasal obstruction except in the very late stages.

II. THE STRUCTURES INVADED BY THE PRIMARY GROWTH.

This part of the study of nasopharyngeal tumours is of great interest. By virtue of their anatomical situation these tumours are capable of producing a series of very definite signs while the actual extent of the growth is very small. Also, their habit of spreading widely along submucous and subfascial planes, instead of forming a large mass in the situation of their origin, leads to an involvement of muscles of a very much more definite type than is common in other growths of the mouth and pharynx, and tends to produce a more widespread involvement of nerves, e.g., the intracranial involvement of the 6th nerve.

The structures which may be involved by the tumour may be classified as follows: (1) *Nerves*: the trigeminal nerve, the 6th nerve, the nerves of the palate, other cranial nerves. (2) *Muscles*: the internal pterygoid, the levator palati. (3) *The eustachian tube.* (4) *The cranium.*

1. INVOLVEMENT OF NERVES.

The Trigeminal Nerve.—

First Division.—Involvement of the first division of the trigeminal nerve has not occurred within my experience, and I am not aware that it has been recorded. This is not surprising, as involvement could only occur either by an extensive intracranial spread of the tumour, or by an invasion of the orbit itself, neither of which events is common.

Second Division.—Involvement of the second division is common in the early stages of the growth of the tumour. The symptom complained of is usually 'toothache', and three of the patients in my small series had had teeth extracted on account of toothache before their admission to hospital. As this symptom occurs early and is seldom or never associated with involvement of the first division, it is presumably due to an extracranial involvement of the second division. This probably takes place in the pterygopalatine fossa, a space which is accessible to the growth as it spreads forward on the base of the skull. An alternative possibility, as the pain complained of is usually in the molar teeth, is that the posterior alveolar branches of the nerve are involved as they pass over the tuberosity of the maxilla. Paræsthesia and anæsthesia in the distribution of the maxillary nerve may occur in the later stages, but its absence, even after the pain has persisted for many months, cannot be taken as evidence of absence of a growth.

Third Division.—Involvement of this division is very common, and may almost be said to be a characteristic part of the syndrome. Here again the pain is often of the character of 'toothache', for which teeth may be extracted. Sometimes, however, pain in the tongue (*Case* 1) or on the face is complained of. It is in the distribution of the third division that anæsthesia most frequently occurs. It is curious that the anæsthesia is often most definite in the distribution of the mental branch of the inferior alveolar nerve. I am unable to offer any explanation of this interesting phenomenon, and it does not seem likely that it can be in any way dependent on the confinement of the inferior alveolar nerve within its bony canal, as there is no evidence that the growth in its early stages approaches anywhere near to the canal.

Involvement of the motor division of the mandibular nerve may occur, and results in paralysis of the muscles of mastication on the side of the growth. This condition may be most easily recognized by the deviation of the point of the jaw to the paralysed side which occurs on opening the mouth. This is produced by the unopposed contraction of the external pterygoid muscle on the non-paralysed side. Palpation of the masseter on the paralysed side may be deceptive, as the rotary movement produced by the single external pterygoid turns the ramus of the jaw on a vertical axis, and this movement when felt on the paralysed side may easily be mistaken for a contraction of the masseter. Palpation of the temporal muscles provides an unmistakable physical sign.

The Sixth Nerve.—The 6th nerve is occasionally involved, usually late in the course of the disease, although in one of the cases here recorded it was the first sign of involvement of the nervous system. It is a little difficult to account for this feature of the growth, but I think most probably the nerve is attacked either directly or, more probably, by an interference with its vascular supply, in the short interval between the point at which it pierces the dura mater and that at which it enters the cavernous sinus. It lies here on the dorsum sellæ, and would be easily attacked by an upward extension of the growth either through the carotid canal or directly through the sphenoidal sinus. That extension in this direction does occur is shown by a radiogram of one of my cases (*Case* 1) in which erosion of the dorsum sellæ is seen to have taken place, and also by the post-mortem findings in *Case* 2.

The Nerves of the Palate.—A proportion of cases shows involvement of the palate other than by direct extension of the growth. The proportion in the cases here recorded is small (two in nine), but paralysis of the palate is regarded by some as one of the cardinal signs of endothelioma of the naso-pharynx and consequently must be considered. It has been held (Trotter[1]) that the paralysis of the palate is due to infiltration of the levator palati as a result of a direct extension of the growth. This appears to me improbable, as in this case one would expect a condition analogous to the trismus which arises from infiltration of the internal pterygoid—that is to say, a hardening and shortening of the muscle, producing a *raised* and immobile palate. This condition I have never seen, nor is it mentioned in any of the case records which I have consulted. Further, in the case in my series which showed paralysis of the palate there was also anæsthesia of the soft palate—no amount of manipulation of the soft palate causing the patient the slightest discomfort. In the later stages of the disease the soft palate may be rendered immobile by direct infiltration of the growth.

The association of paralysis of the soft palate with anæsthesia is difficult to explain if one accepts in its entirety the work of Aldren Turner[2] on the innervation of the soft palate. It is generally agreed that the sensory innervation of the soft palate is obtained from Meckel's ganglion. Previous to the work of Aldren Turner it was held that the muscles of the palate, with the exception of the tensor palati, were supplied by the facial nerve through Meckel's ganglion, while the tensor was supplied by the motor division of the 5th nerve through the otic ganglion. Turner's work gives good evidence that the nucleus concerned is not that of the facial nerve, but those of the vagus and accessory; the evidence concerning the subsequent course of the fibres, however, is not so clear.

Turner's statement that "the muscles of the soft palate derive their motor supply from the pharyngeal plexus through the pharyngeal branch of the vagus" is supported only by the results of experiments on dogs and by one case recorded by Dr. Bastian,[3] in which excision of Meckel's ganglion was performed without giving rise to any palatal paralysis. More recent work by A. R. Rich[4] suggests that the tensor palati is really supplied by the 5th nerve. According to this author, paralysis of the tensor gives rise to no deformity of the palate, and can be recognized only after stripping the mucous membrane from the muscles. He has performed this operation on dogs and has observed paralysis of the tensor palati after section of the mandibular division of the 5th nerve. If Rich's work is accepted, the clinical 'paralysis of the palate' is simply a paralysis of the levator palati. As regards the origin of the nerve-supply of this muscle, he agrees with Turner in attributing it to the 11th nucleus, but again gives no convincing account of the subsequent course of the fibres.

There are several circumstances which suggest to me that the course is not through the pharyngeal branch of the vagus nerve. One of these is the coincidence of anæsthesia with paralysis of the soft palate, and another the much more frequent occurrence of paralysis of the palate than of signs pointing to involvement of the 10th and 11th nerves (cf. Woltman's table in the abstract of the literature—p. 260—in which the incidence of palatal paralysis

is exactly that of involvement of the motor division of the 5th nerve and much greater than that of involvement of the 11th nerve).

A possible course for the fibres between the 11th nucleus and the levator palati is through the jugular ganglion of the vagus and thence by the auricular nerve to the facial nerve. The further course would then be by way of the greater superficial petrosal nerve and the nerve of the pterygoid canal to Meckel's ganglion. This is a suggestion admittedly quite unsupported by experimental evidence, but it furnishes a reasonable explanation of the coincidence of anæsthesia and paralysis of the soft palate in the type of case which I am describing, and renders understandable the traditional association of paralysis of the palate with facial paralysis.

Since this article was written, Mr. Julian Taylor, of the staff of University College Hospital, has very kindly drawn my attention to a case which came under his care a short time ago and which appears to have an important bearing on the question of the course of motor nerves to the soft palate. The case history was as follows :—

A man, age 26, was admitted to hospital fourteen weeks after a motor-cycle accident. At the time of the accident there had been definite concussion, with retrograde amnesia, and, according to the statement of the patient, bleeding from the right ear and the throat. There was also swelling of the right eye. On admission the patient showed the following nerve lesions, all of which were on the right side.

Second Nerve.—Complete loss of sight in the right eye, with loss of direct (not consensual) pupillary reaction.

Fifth Nerve.—Motor : Paralysis of the muscles of mastication. Sensory : Diminution of sensory acuity and parasthesia in the distribution of the second and third divisions.

Chorda Tympani.—Perception of taste on right half of tongue delayed and incomplete ; ordinary sensation over tongue normal.

Palatal Nerves.—" When the palate is raised the uvula is seen to be displaced upwards and to the left with flattening of the right half of the arch."

The rest of the cranial nerves showed no impairment of function. In the radiogram a crack could be seen in the right parietal bone.

Here, then, is a patient who has certainly had a fracture in the middle fossa of the skull on the right side. There is no evidence whatsoever of any lesion in the posterior fossa. He shows among the cranial nerve lesions a paralysis of the soft palate, apparently of the levator palati—I say this because in a very full and accurate description of the case there is no mention of deformity of the palate at rest. It is almost inconceivable that the lesion of the palatal nerves can have occurred at any other place than in relation to the base of the middle fossa of the skull. A study of the anatomy of the middle fossa of the skull shows that the hiatus of the facial canal (from which issues the greater superficial petrosal nerve) lies immediately above the inner end of the petrotympanic fissure (through which the chorda tympani leaves the skull), and that both are in close relation on their inner side to the motor division and second and third sensory divisions of the 5th nerve.

I regard this case as evidence in favour of the view that the motor nerve supply of the levator palati is carried by the greater superficial petrosal nerve, and as showing practically beyond doubt that, whatever may be the exact course of the fibres, they are in relation to some part of the middle fossa of the skull and not derived " from the pharyngeal plexus through the pharyngeal branch of the vagus."

Other Cranial Nerves.—Reference to the review of the literature at the end of this article will show that there is no cranial nerve which has not been observed to be involved in the extension of a malignant nasopharyngeal

growth. This observation is not of any value for my present purpose, owing to the fact that in these records the various types of nasopharyngeal tumour are not well distinguished. It is clear that in the terminal stages of the endothelioma there may be extension of the growth so as to invade the nerves of the jugular foramen. This was present in one of my cases, roughly nine months after the onset of the disease, which in this case ran a particularly rapid course. Paralysis of the sympathetic on the side of the tumour was also present, although there were no palpable glands in the neck. The unfortunate patient was in a truly pitiable condition, being unable to hear owing to involvement of the Eustachian tubes, and unable to speak or swallow from paralysis of the muscles of the larynx and pharynx. In this case paralysis of the pharyngeal muscles was responsible ultimately for the death of the patient from inanition.

It is also beyond doubt that an intracranial spread of the tumour, without being extensive, can involve other nerves in the anterior part of the middle fossa. In this way the 2nd, 3rd, and 4th cranial nerves may be involved, but such an event is not common. It has occurred once only in my own experience, and this at a very late stage. However, in view of the occasional early involvement of the 6th nerve, which I believe is due to a direct invasion either of the nerve or of its blood-supply in the immediate neighbourhood, it is not impossible that cases may arise in which the spread of the growth occurs in such a manner as to lead to the early involvement of the 2nd, 3rd, and 4th nerves.

2. INVASION OF MUSCLES.

The Internal Pterygoid.—This muscle may be infiltrated directly by the growth. When this involvement occurs it leads to trismus of a greater or less degree. Trismus does not necessarily occur even in the late stages of the disease, and as far as my own cases are concerned its presence was only observed in three. On the other hand, in *Case* 2 trismus was the earliest sign referable to the growth as distinguished from its metastases.

The Levator Palati.—I have already stated my reasons for believing that direct involvement of this muscle is rare. None of my cases showed the physical signs which one would expect if this muscle were infiltrated by growth. It is, however, recorded by Trotter.

3. INVOLVEMENT OF THE EUSTACHIAN TUBE.

The growth originates close to the pharyngeal opening of the Eustachian tube, and commonly causes, sooner or later, an obstruction of the lumen of the tube. This leads first to deafness on the side affected, and sometimes later to an otitis media. Unilateral deafness is a very characteristic early sign of the disease. It is only recorded in four of my cases, but I think this gives a false impression of its frequency, as it is a sign which may easily be overlooked when the patient has other more prominent signs. There is, however, one recorded case in which deafness was looked for and found to be absent.

Otitis media may result from continued obstruction to the Eustachian

tube. It was present in the later stages of one of my own cases, and was observed in one of Trotter's cases in which the growth recurred after removal. It is also mentioned by New.[5]

4. INVASION OF THE CRANIAL CAVITY.

This has been recorded fairly frequently. Involvement of the 6th nerve is, as has already been stated, probably due to an intracranial extension of the growth; also the radiogram of the skull in one of my own cases showed erosion of the dorsum sellæ. Gross clinical evidence of intracranial invasion is not, however, common. Reverchon[6] refers to it and to relief of the accompanying symptoms by lumbar puncture and subtemporal decompression. New refers to the possibility of an intracranial extension of one of these tumours being mistaken for a primary endothelioma of the Gasserian ganglion. This mistake has apparently occurred when the ganglion has been exposed for a supposed 'major neuralgia' of the 5th nerve. The error does not appear to be a likely one, as the clinical signs concerned with the 5th nerve in true 'trigeminal neuralgia' are very different from those which are seen in endothelioma of the nasopharynx. Intracranial extension does occur, and should be considered in estimating the suitability of any particular case for treatment; but none of the cases I have seen, even in their terminal stages, showed any gross clinical sign of intracranial involvement beyond the paralysis of cranial nerves. *Case* 1, when seen shortly before his death, had no headache and no sign of intracranial extension, although the radiogram taken five months previously showed erosion of the dorsum sellæ. Post-mortem evidence is regrettably scarce, but in the few available records intracranial extension is frequently mentioned. Probably before death most of these tumours do invade the cranial cavity, but it must be emphasized that the signs of a cerebral tumour or even of increased intracranial pressure have no place in the clinical picture of the cases with which I am dealing. I mention this particularly as there is a tendency among the French authors to speak of these growths as if intracranial invasion were an early and prominent feature, whereas in the experience of others, although the intracranial invasion may take place early, it gives rise to physical signs either very late or never.

III. THE CLINICAL CHARACTERISTICS OF THE GLANDS INVADED BY SECONDARY DEPOSITS.

This part of the study of these tumours is both interesting and important to the clinician. As Trotter has pointed out, the type of glandular enlargement is highly characteristic, so much so that to one who is familiar with the condition the discovery of glands of this type in a young person is sufficient to suggest the site of the primary growth. The importance of being able to recognize these glands lies in the fact that glandular enlargement may occur before the primary growth has given rise to any gross physical signs.

The glands involved are those of the upper deep cervical group, and frequently invasion on both sides of the neck is simultaneous, or practically so. The site of the earliest invasion is, unfortunately, not characteristic;

commonly the largest glands, and presumably the earliest affected, are those lying on the jugular vein behind the angle of the jaw. From here the invasion extends to affect the whole deep cervical group, and ultimately sometimes the glands of the posterior triangle. Invasion of the retropharyngeal glands appears to be rare; none of my cases showed any sign suggesting such an invasion, neither is it mentioned in any operation notes or post-mortem record which I have consulted. Neither has invasion of the glands in the parotid region occurred in my experience, nor apparently in that of other writers on the subject. Consequently the distribution of the glands invaded is different from that found in malignant disease in other parts of the pharynx and mouth only in that the invasion is commonly bilateral in the early stages and may occur before the primary growth has given rise to any symptoms. Such a combination of signs in a young patient may easily lead to a diagnosis of tuberculous disease or lymphadenoma.

It is accordingly necessary to lay some stress on the character of the enlargement, which is, in my opinion, more helpful than the distribution. The glands are extremely hard, and very early become fixed to surrounding structures. Yet at the same time they remain distinct from one another, and the fixity is rather of the type produced by a widespread inflammatory reaction outside the capsule of the gland than of growth extending through the capsule. The impression produced on the examining hand is that of a number of hard glands fixed together by a softer fibrous mass. The glands are frequently tender to palpation to a greater degree than carcinomatous glands at the same stage of involvement.

In Trotter's paper the clinical appearance of the glands is compared with that produced by tuberculous disease, and, apart from the fact that the endotheliomatous glands are originally harder and have no tendency towards breaking down, the comparison is excellent.

IV. REMOTE METASTASES.

Conclusions on this subject are only justifiable if one has at one's disposal the records of a large number of cases observed up to their conclusion and with post-mortem records. A few cases of malignant nasopharyngeal growths showing remote metastases are recorded in the literature of the subject,[7, 11, 12] but none of these cases, so far as I can judge, belonged to the endothelioma group. In my own series two cases showed evidence of remote metastases. One patient (*Case* 4) had, when last seen, eighteen months after the onset of the disease, a large mass in the liver, the mass having appeared within the space of a few months. The other patient (*Case* 2), when seen fourteen months after the onset, was suffering with pain in the back and legs, the distribution of which strongly suggested the presence of pressure on the spinal nerve roots in the lumbar region. The radiogram of the spine showed no definite evidence of growth and no collapse of the vertebræ, but I have very little doubt that a secondary growth was present.*

These two examples of secondary growths occurring in a series of five

---

* Numerous general metastases were found at the post-mortem in this case.

cases, only four of which were observed in their terminal stages, suggest that the occurrence of remote metastases is fairly frequent. It does not, however, appear to be an early event in the history of the disease. It remains to be seen to what extent remote metastases would occur if the primary growth were eradicated by the use of radium.

V. THE EARLY SYMPTOMS OF THE GROWTH.

In judging the early symptoms of the growth, I have taken into consideration, in addition to the 5 cases which have come under my personal observation, 4 cases which were undoubted examples of the disease recorded in the case books of Mr. Bilton Pollard and Mr. Wilfred Trotter. Probably many more could have been found, but I have confined myself, in speaking of cases which have not come under my personal observation, to those which were undoubted examples of the condition in question and in which the notes are reasonably complete. Using these 9 cases, the initial symptoms were as follows: enlargement of the glands of the neck, 3 cases; pain in the distribution of the 5th nerve, 3 cases; deafness, 2 cases; hæmorrhage from the pharynx, 1 case.

It is interesting to note that in all three cases which showed pain in the distribution of the 5th nerve as an original symptom, attempts had been made to relieve the pain by extraction of teeth. In two cases teeth were extracted from the upper jaw, in the third the actual teeth extracted are not mentioned.

In order to give some idea of the frequency of the various symptoms and signs which I have mentioned, I have prepared a table (*Table I*, p. 252) showing the early signs in the 9 cases which I am here considering.

VI. THE MICROSCOPIC APPEARANCES OF THE GROWTH.

This is not a matter with which I propose to deal in detail: firstly, because I do not feel competent to deal arbitrarily with a matter in which pathologists find themselves at variance; and, secondly, because among the sections at my disposal the indifferent and poor greatly outnumber the good.

It does not appear to me to be of great importance whether the tumour is called an endothelioma or is grouped as a special form of carcinoma. The sections which I have at my disposal are derived from six of the nine cases I have quoted. All these sections both from the primary growths and from the glandular metastases show a sufficient number of points of similarity to justify their inclusion in a single group.

The growth at first sight strongly suggests a carcinoma, as it is composed of large irregular cells, mostly spheroidal, lying in groups which are separated by a considerable amount of connective tissue. Throughout the tumour there is no sign of degeneration and there are no cell nests. On closer inspection there is a very evident tendency for the cells to be arranged in relation to clear spaces which do not appear to be the result of degeneration. In some sections the spaces show as fairly large clefts, but a commoner appearance is for the cells to be arranged so as to form tubules, the diameter of which is only two or three times that of a single cell. This appearance is well shown in the microphotograph reproduced from the section of the tumour

Table I.—Frequency of the Cardinal Early Symptoms in Nine Cases of Endothelioma of the Nasopharynx.

Case	Enlargement of Cervical Glands	Pain in Distribution of 5th Nerve	Anæsthesia or Paralysis in Distribution of 5th Nerve	Swelling in Mouth*	Trismus	Hæmorrhage from Nose or Pharynx	Palatal Paralysis	Unilateral Deafness	Time since Onset	Notes
1. F. G. Surgical Unit 1928	No	Yes Teeth extracted	Yes	No	Slight	Yes	Yes	Yes	3 months	—
2. J. M. Surgical Unit 1928	Yes	Yes	No	No	Yes	No	No	No	3 to 4 months	—
3. E. W. Mr. Williams 1925 and 1926	Yes	No	No	No	No	No	No	Yes	4 months	Notes state that palate was 'bulged down'
4. A. R. Mr. E. K. Martin 1926	No	Yes Teeth extracted	No	Yes	No	Yes	No	No	15 months	—
5. H. B. Surgical Unit 1924	No	Yes Teeth extracted	No	Yes	No	No	No	No	6 weeks (probably more)	—
6. C. Mr. Trotter 1919	Yes	Yes	Paræs-thesia	No	No	Yes	Yes	Yes	5 months	—
7. P. Mr. Pollard 1913	Yes	No	No	No	No	No	No	No	3 to 4 months	Notes state that palate was 'bulged'
8. J. Mr. Trotter 1911	No	No	No	No	No	Yes	No	No	5 months	Notes incomplete
9. B. Mr. Trotter 1912	Yes	Yes	No	No	Yes	No	No	Yes	2½ years	Discharge from ear
Total of Positive Cases	5	6	1	2	3	4	2	4		

* By swelling in the mouth I mean a swelling below the level of the palate which can be seen without post-nasal examination.

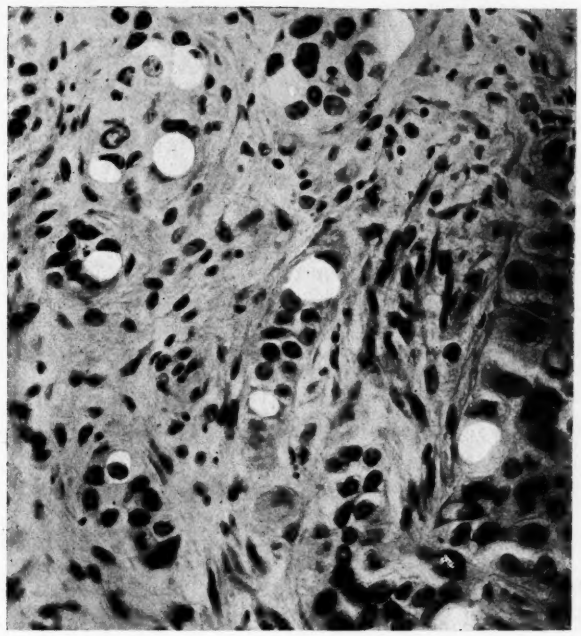


FIG. 156.—*Case* 5. The tubular arrangement of the cells is well seen, particularly in the centre of the figure, where a curved tubule has been divided transversely at both ends, the intermediate part of the section running through the wall of the tubule. ($\frac{1}{6}$ obj.)

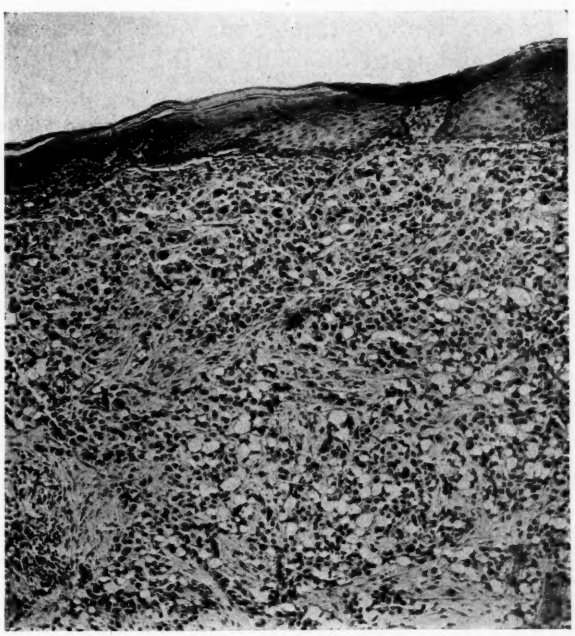


FIG. 157.—Showing the general structure of the tumour and its tendency to submucous spread. ($\frac{2}{3}$ obj.)

taken from *Case* 5 (*Fig.* 156). The section here has run through the curved part of a U-shaped tubule, dividing the tubule more or less transversely at both ends and running through its wall in the intermediate part. The arrangement is by no means as regular as this in all the sections or in all parts of the same section. In many places the shape of the cells can be seen to be adapted to the formation of a regular lumen. Another striking feature of the growth, which is in accord with its clinical behaviour, is that, however close the tumour substance approaches to the mucous membrane, it has no effect on it apart from what appears to be a pressure atrophy over the most prominent part of the growth. This peculiarity is well shown in *Fig.* 157, where the epithelium of the mucous membrane can be seen to be thinned by pressure to a marked degree without being actually invaded.

I am hopeful that in course of time, as these tumours become better recognized, it will be possible to discover the exact point of origin of the growth, and possibly in the hands of an expert pathologist the peculiar microscopic structure might furnish some clue.

VII. POSSIBLE LINES OF TREATMENT.

A very cursory consideration of the physical signs of these tumours will be sufficient to convince the majority of surgeons of the futility of ordinary surgical methods as a means of treatment. Trotter has obtained a temporary success in one of his cases, the patient being free from recurrence for eighteen months. Considering the manner in which these tumours spread, and the situation in which they originate, such a result is sufficiently gratifying, but it cannot be said to be satisfactory. The only case in my own series in which the question of operability arose was *Case* 1, and in view of the fact that both the second and third divisions of the 5th nerve were involved it was not considered that an attempt at radical removal could be justified.

The question of treatment by radium has yet to be considered. *Cases* 2 and 3 of my series were treated with radium for the enlarged cervical glands, in both cases with some measure of success; *Case* 3 is at the time of writing still under my observation, and the glands have progressed but little since the time at which I first saw him some nine months ago. The only cases in which a direct application of radium to the growth was tried are *Cases* 4 and 5, where little or no improvement followed. Here the growth had extended very widely before any treatment was attempted, and the irradiated part of the tumour can only have been the outlying margin of a very extensive mass. However, the fact that the glands can be kept in check by radium seems to show that radium has some influence on the tumour. The only line of treatment which appears to me to offer any hope at present is the insertion of seedling tubes of radium emanation in the directions in which we know the tumour to spread. This can, I think, be done only by exposing the tumour by means of Trotter's osteoplastic resection of the upper jaw; I have made one attempt at inserting radium without exposing the tumour, and was impressed by the obvious futility of the proceeding.

The outlook from the point of view of treatment is distinctly gloomy, but I should not now hesitate to use seedling radon tubes after exposing the

growth. As a purely palliative measure where the pain is severe an alcohol injection of the Gasserian ganglion may be tried. In the only case where I have done this there was a definite but transient relief.

CASE RECORDS.

The cases here recorded are those which have come under my personal observation. That the clinical observations in the earlier cases are not more complete is due to the fact that I did not at the time realize that they belonged to this group and consequently did not look for signs such as unilateral deafness. There is, however, in my opinion, sufficient evidence to justify grouping these five cases together as examples of endothelioma of the nasopharynx in its different stages and different aspects.

Case 1.—(Surgical Unit 4955, 1928). F. G., a man of 56, was admitted with a three months' history of pain in the left side of the face extending from the eye to the lower border of the mandible. Carious teeth had been removed without influencing the pain. During the three months the patient had noticed the onset of deafness on the left side and difficulty in opening his mouth. For one month there had been an occasional burning sensation on the left side of the tongue. A few days before admission he had a profuse hæmorrhage from his nose and mouth.

Physical Signs—A strong healthy-looking man. The speech suggested obstruction of the nose. There was pain in distribution of second and third divisions of the 5th nerve on left side; paræsthesia and numbness in the distribution of the second division; anæsthesia over area supplied by mental branch; paralysis of muscles of mastication on left side. The soft palate was insensitive and immobile on left side, and there was deafness in the left ear. A hard sessile mass was palpable on the left lateral wall of the nasopharynx; it extended down to the tonsil, and was not ulcerated. The X-ray showed erosion of the dorsum sellæ. There was no enlargement of glands in neck. No treatment was attempted.

Further Progress.—This was rapid, and five months after admission the patient was moribund from inanition following on paralysis of the pharyngeal muscles. There was also paralysis of the larynx, and the left sympathetic was involved. There were no obvious secondary deposits, no enlarged glands, and no signs of intracranial involvement.

Remarks.—This case may be taken as typical of the condition, apart from the absence of glandular involvement.

Case 2.—(Surgical Unit 4808 and 4939, 1928). J. M., a man of 29, had had swelling in left side of neck for three or four months, stiffness of the jaw for five or six weeks, aching pain in gums of left side for two weeks.

On Examination.—He was fairly well nourished. There was marked trismus. Typical 'endotheliomatous' glands were noted in the left deep cervical group and one large gland on right side. No anæsthesia in distribution of the 5th nerve. X rays showed a wisdom tooth lying horizontally in the lower jaw on the left side. The nasopharynx was not examined, owing to trismus.

Treatment.—The wisdom tooth was removed, and a gland excised for section. Subsequently numerous applications of radium were made to the glands in the neck, and an alcohol injection of the Gasserian ganglion was attempted for relief of pain. This was temporarily successful.

Microscopic Section of the gland shows a typical endothelioma.

Progress.—Nine months after the onset a firm submucous swelling appeared at the junction of the hard and soft palate on the left side. A month later a mass appeared filling up the left temporal fossa (*see Fig.* 155). There was then a left external rectus palsy and anæsthesia over the distribution of the mental branch of the trigeminal. The patient complained of a discharge from the left ear and

deafness. No palatal paralysis. The glands had increased somewhat in size, but were kept in check by applications of radium. His general nourishment remained good.

Five months later, and fourteen months after the onset of the disease, this patient was readmitted in a very wasted condition. The 6th-nerve paralysis had then disappeared and subsequently varied in degree from day to day, but the left pupil showed a deficient reaction to light and accommodation, presumably as a result of a partial paralysis of the 3rd nerve. The patient also complained of dimness of vision in the left eye, and this eye subsequently became almost blind. There was nothing to suggest the presence of growth in the orbit, and on examination of the optic disc there was no optic neuritis. There was some difficulty in swallowing, which appeared to be due neither to pain nor to mechanical obstruction. Headache was absent, and the pain in the face was still strictly limited to the left side.

The glands had increased greatly in size during the previous month, and the patient complained of pain in the lumbar spine, girdle pain, and pain in the front of the legs. There was no deformity of the spine, and X rays failed to show any definite evidence of invasion of the lumbar vertebræ.

The patient gradually became weaker, and died, about sixteen months after the onset of the disease, from pneumonia.

Post-mortem Examination.—Thanks to the skill of Dr. Barnard, who performed the post-mortem examination and removed the specimen, I was able to examine the relation of the growth to the base of the skull in some detail. The specimen as it came to me consisted of a large portion of the central part of the base of the skull, together with the maxillæ and nasal cavities, the pharynx, tongue, larynx, and the great vessels of the neck.

From this specimen I removed the basiocciput, and the body and small wings of the sphenoid bone. The bone here showed no sign of invasion by growth, and both the sphenoidal sinus and the pituitary fossa were free from involvement. The remaining portions of the great wing of the sphenoid were removed piecemeal to permit of tracing the branches of the 5th nerve into the growth. The growth was firmly attached to the bone in the neighbourhood of the foramen ovale, and the bone here appeared to have been eroded. The microscopic section of the bone, however, showed no actual invasion by growth. The specimen was then bisected in a sagittal plane, leaving the inner end of the petrous part of the temporal bone *in situ* on both sides.

On examining the specimen from inside the pharynx there is no ulceration of the mucous membrane. The left Eustachian orifice is obscured by a submucous swelling, and the whole lumen of the nasopharynx on the left side is diminished, although the pharynx is held permanently open by the rigidity of its walls on the left side, giving the lumen, in cross-section, a wedge-shaped appearance with the base of the wedge to the left. Examination of the divided wall of the pharynx shows that its roof is infiltrated with growth, as also is the soft palate. The growth has not extended forward so as to obstruct the nasal choanæ.

Examination from outside shows a mass of growth on the left lateral wall of the pharynx, continuous below with a glandular mass which surrounds the great vessels of the neck. The mass occupies the pterygoid region and, extending forward, has completely filled the maxillary antrum, encircling and destroying the pterygoid plates and the posterior wall of the antrum. The growth partially encircles the ramus of the lower jaw.

On the roof of the pharynx, in the situation from which the basisphenoid has been removed, is a mass of growth more than a centimetre in thickness in its hardened state. It extends across the middle line as far as the right foramen ovale.

The sole intracranial spread of the tumour is by way of the carotid canal. On the left side the carotid artery is embedded in a process of the growth which extends up into the interior of the skull, but apparently stops short of the cavernous sinus. The left 6th nerve as it passes over the lip of the petrous temporal enters this process of growth. The same process of growth turns outwards over the petrous

temporal and runs beneath the Gasserian ganglion. The first and second divisions of the 5th nerve pass forward without being obviously involved in the growth, but the third division passes downwards through the foramen ovale into a mass of growth outside the skull.

The 4th and 3rd nerves can be traced in the cavernous sinus and are apparently free from growth, although they are less easy to trace than on the right side. There is no macroscopic involvement of the optic nerve.

The carotid artery is not thrombosed. The whole extent of the intracranial growth is not greater than that of the Gasserian ganglion. On the right side the growth has invaded the lower end of the carotid canal, but has not reached the interior of the skull and has not involved the 6th nerve.

Metastases having the same macroscopic appearance as the primary growth were found in several ribs, in the spine, and in the left lobe of the liver.

The macroscopic evidence suggests that the 6th nerve and the third and second divisions of the 5th nerve were involved directly in the growth. The cause of the disturbance of the function of the oculomotor and optic nerves is less obvious, but was in all probability an interference with their blood-supply. In the earlier stages this was probably true also of the 6th nerve, as the loss of function was temporary at first and tended to vary in degree for some time.

Microscopic sections were made of the optic nerve proximal to the orbit, and of the 3rd nerve and the first and second divisions of the 5th in the cavernous sinus. The optic nerve showed no invasion by growth and was not degenerated at the point where the section was made; hence the involvement was probably posterior to the section, i.e., intracranial and not intra-orbital. The sections of the 3rd nerve and the first and second divisions of the 5th, taken from these nerves in the cavernous sinus, showed no growth.

The post-mortem is interesting mainly in showing the small extent of the intracranial spread in relation to the size of the extracranial mass, and in demonstrating that clinical signs of interference with the function of the nerves running in the cavernous sinus may be present before the growth has actually reached the nerves in question.

Remarks.—The nasopharynx was never examined in this case owing to the trismus. The symptoms and the microscopic appearances, in association with the appearance of the swelling in the mouth, left no doubt as to the nature of the tumour. Radium was temporarily successful in keeping the progress in the glands in check.

Case 3.—(Mr. Gwynne Williams, 4611, 1925, and 834, 1926.) E. W., a woman of 44, had complained of lumps in the left side of the neck for four months, and similar lumps on the right side for one month. Previous to this she had become deaf in the left ear.

On Examination.—She was fairly well nourished. There were typical 'endotheliomatous' glands on both sides of the neck, and by the mouth was a hard mass which bulged down the soft palate and could be felt on the left lateral wall of the nasopharynx. She had no pain except on palpation of the glands.

Microscopic Section of Tumour.—Endothelioma.

Treatment.—Radium was inserted into the glands and the primary tumour.

Progress.—The glands diminished in size. Two months after the first application an external rectus palsy appeared on the left side. A further application of radium was made to the glands. The patient was not seen again.

Remarks.—These tumours are unusual in women. The absence of pain is a remarkable feature. On her second admission the patient complained of loss of voice, which may have been due to a commencing laryngeal paralysis.

Case 4.—(Mr. E. K. Martin, 2936 and 3855, 1928.) A. R., male, age 58, fifteen months previous to admission had a copious hæmorrhage from his mouth which lasted for one night. Previous to this he had had 'toothache' for which some teeth had been extracted. He was then fairly well for a year, after which a recurrence of the toothache was followed by a further hæmorrhage.

PHYSICAL SIGNS.—A pale but well-nourished man. There is a firm submucous swelling extending forward from the junction of the hard and soft palate to the incisor region. The palatal swelling is more marked on the left side, and there is a visible swelling of the face over the left maxilla. There are no enlarged glands in the neck. There is some nasal obstruction, which the patient believes to be more marked on the left side.

MICROSCOPIC SECTION OF TUMOUR.—Endothelioma.

TREATMENT.—Radium was applied to the accessible parts of the tumour without producing any marked improvement.

PROGRESS.—Three months later the patient was readmitted. He was then obviously wasted. The tumour had increased in size by a further submucous extension, so that the upper jaw and alveolus on both sides were enormously enlarged. There was an ulcer at the junction of the hard and soft palate on the left side. There was a large mass in the liver. A further application of radium produced no beneficial result.

REMARKS.—There is perhaps some doubt as to whether this case is rightly included in the series. There was, however, a long period between the onset of symptoms referable to the presence of a tumour and the appearance of the tumour in the mouth. When the growth did appear it was in the typical situation and showed the typical submucous spread. Microscopically it coincided with those of the other cases in the series. In combination with the presence of 'toothache' as an original symptom, these facts appear to me to mark this case as an endothelioma of the nasopharynx.

Case 5.—(Surgical Unit 2590 and 2710, 1924.) H. B., a man of 59, was admitted complaining of pain in the right upper and lower jaw, following extraction of carious teeth, and swelling of the face. The teeth had been removed six weeks before admission. He was a thin man. There was a visible swelling of the right side of the face. A week later a submucous swelling appeared at the junction of the hard and soft palate on the right side. Six weeks later there was a large swelling in the mouth on the right side, extending over the alveolus. The mass was superficially ulcerated. The palate on the right side was pushed down and immobile, and the uvula was pushed over to the left. The right temporal fossa was filled up by a mass beneath the deep fascia, producing an obvious deformity of the face. There were no enlarged glands in the neck.

MICROSCOPIC SECTION.—Endothelioma (*see Fig.* 156).

TREATMENT.—Radium was inserted into the accessible part of the tumour. This produced a very temporary improvement, and the patient was shortly afterwards transferred to an infirmary.

REMARKS.—I think there can be no doubt that this case belongs to the group of tumours in question. The onset with pain in the distribution of the 5th nerve, the site of appearance and submucous character of the tumour in the mouth, and the microscopic appearance of the tumour, are sufficient to justify its inclusion. In addition to these points, the extension of the tumour into the temporal fossa took place in exactly the same manner as that in *Case* 2. There are no observations as to deafness and anæsthesia in the distribution of the 5th nerve, as I saw the case before I was familiar with the numerous and diverse manifestations of these tumours, and did not at the time realize that this case belonged to the group.

LITERATURE OF THE SUBJECT.

For several reasons I have thought it advisable to devote a separate section of this article to a review of the literature of the subject. Reports of malignant tumours of the nasopharynx showing involvement of the nervous system are fairly numerous, but as a result of the diversity of the symptoms, cases have been reported by neurologists, laryngologists, pathologists, and ophthalmologists, and naturally each specialist has tended to dwell on the

aspects of the case most interesting to himself. It is clear that many of the reported cases belong to the group which I have attempted to describe, but often examples of other forms of tumour are included under the same heading. A further difficulty is introduced by the fact that there is no uniformity of opinion as to the interpretation of the microscopic appearances of the endothelioma, and it is usually impossible to gather from the reports of microscopic examination whether or not a particular reported case belongs to this group. In a few cases clinical peculiarities of the tumour sufficient to place it are mentioned, but more often one finds the unadorned statement that a tumour was present in the nasopharynx.

The term 'endothelioma' is admittedly more or less properly applied to 'mixed' or 'salivary' tumours, and some reported cases of endotheliomata in the pharyngeal region are clearly examples of these tumours, which are met with fairly frequently in the palate.[8] They possess no points in common with the endothelioma of the nasopharynx beyond their somewhat unfortunate name.

The earliest reference which I have consulted is a long article published in 1907 by Harmer and Glas.[9] This article is concerned mainly with tumours originating within the nasal cavity, sometimes invading the cranial cavity through the cribriform plate, and the orbit in the neighbourhood of the lamina papyracea. These tumours are naturally liable to be associated with invasion of the first four cranial nerves, and may also give rise to neuralgic pain. The pain, however, is often in the distribution of the first division of the trigeminal nerve, sometimes in that of the second division, and (as far as I am able to judge from the literature) never in that of the third. Moreover, tumours of this type are liable to be associated quite early in their course with proptosis, which is due to actual filling up of the orbit with growth, and with definite symptoms pointing to a nasal origin, such as epistaxis and nasal obstruction. The pain appears to be due in many cases to obstruction of the outlet of the frontal sinus or the maxillary antrum, and suppuration may arise in these cavities from the same cause. Naturally these tumours do not often come under the care of a general surgeon, and, consequently, my knowledge of them is gathered almost entirely from the literature of the subject. It appears certain to me that much of the difficulty which one encounters in attempting to form a clear conception of malignant tumours of the nasopharynx from lists of reported cases is due to the fact that usually no distinction is made between these growths, which are primarily tumours of the nasal cavity with a tendency to secondary involvement of the first four cranial nerves, and the group I have described, which consists of tumours which are really infiltrating growths at the base of the skull accessible to the examining finger in the nasopharynx. Perhaps the most useful distinction is the presence of proptosis in a number of cases of the intranasal type. Harmer and Glas do not classify any of the tumours in their report as endotheliomata, but there was at the time a considerable controversy in Germany as to the characteristics of an endotheliomatous tumour, and the literature of the time tends to place the incidence of the endothelioma very high or very low according to the views of the writer. However, allowing for this point, I have been unable to satisfy myself that any one of the tumours mentioned in this paper belonged to the group which I have described.

A somewhat later article by Pollak,[10] published in 1911, deals almost exclusively with the pathological side of the question, and places the incidence of endotheliomata very much higher.

In 1911, in a paper read before the Medical Society of London, Trotter[1] gave a short account of the tumours with which I am dealing, and it was this paper which first attracted the interest of the present writer to the condition.

Several papers of later date are devoted to an account of nasopharyngeal tumours which have given rise to symptoms of interference with the nervous system, still, however, without any very successful attempt to differentiate the various types. The most valuable of these papers is one published in Chicago in 1922 by H. W. Woltman.[11] In it are reported 25 cases of nasopharyngeal tumours with involvement of the cranial nerves; 15 of these are stated to have been squamous epitheliomata and 6 lymphosarcomata. A list of the nerves which were found to be involved in these 25 cases is, I think, worth quoting fully (*Table II*).

Table II.—Showing the Proportions in which the Cranial Nerves were Involved in 25 Cases of Nasopharyngeal Tumour Reported by H. W. Woltman.

Cranial Nerve	Epitheliomata	All Cases
I	Not determinable	
II	1 in 15	5 in 25
III	2 ,, 15	4 ,, 25
IV	2 ,, 15	4 ,, 25
V Sensory	6 ,, 15	11 ,, 25
V Motor	4 ,, 15	6 ,, 25
VII	1 ,, 15	1 ,, 25
VIII	Not determinable	
Palate	4 in 15	6 in 25
Vocal cords	1 ,, 15	3 ,, 25
XI Spinal	1 ,, 15	3 ,, 25
XII	4 ,, 15	5 ,, 25

In addition to the author's 25 cases a list of cases from the previous literature is given. Some of these tumours belong certainly to the group which I have described; others certainly do not. On the whole, those described as squamous epitheliomata bear a closer resemblance to my cases, and in one of these it is mentioned that in the later stages a swelling appeared in the temporal fossa, presumably of the same nature as that described in *Cases* 2 and 5 of my series.

As regards the question of remote metastases, these were present in one of this author's cases and in three of those which he has collected from the literature. None of these can be said with any certainty to belong to the endothelioma group. Other interesting points are made in this article, particularly that of the possibility of confusion of a malignant nasopharyngeal tumour showing X-ray evidence of destruction of the sella turcica with a pituitary tumour; and that of the varying degree to which the sensations of pain,

touch, and temperature are lost when the 5th nerve is involved in the growth of the tumour.

New,[5] dealing also with the recent cases from the Mayo Clinic, emphasizes the frequency with which patients suffering from malignant nasopharyngeal tumours are submitted to operations such as extraction of teeth or tonsils, in the hope of relieving pain the true cause of which has not been diagnosed.

Crow and Baylor[12] are concerned less with the neurological aspects of the cases than with the treatment by radium. In their article the cases grouped as carcinoma include many which correspond closely to those which I have described. Over one-third of these cases had signs of gross intracranial invasion before death, 13 of 15 had glandular involvement, and 9 had marked trismus. The earliest symptom was pain in the ear or loss of hearing in 7 cases, and glandular enlargement in 3. On the whole, the patients are younger than those of my series, the average age of the 15 cases being about 32. Paralysis of the palate is mentioned in several instances. Radium was used sometimes in the treatment of patients in this group, but no cures are recorded, although a temporary improvement was occasionally produced.

Two interesting cases are excellently reported from the point of view of the ophthalmologist by Stähli, of Zurich.[13] The first of these showed a 6th-nerve palsy as the sole symptom of the condition for a period as long as five months; the second was first seen complaining of a 3rd-nerve palsy. It is possible that the second case was an example of the intranasal type of growth rather than the infiltrating endothelioma. This author has noted the very interesting point that in both these cases the degree of paralysis after the onset was inconstant, and even complete recovery took place for short periods.

There remains to be considered the modern French literature on the subject.

M. Jacod, of Lyons,[14] recognizes the type of growth which I have described, and speaks of it as a 'peritubular sarcoma'. He has devoted much attention to the intracranial spread of these tumours, which he believes to take place through the bony part of the Eustachian tube, whence it is directed forwards into the middle fossa of the skull, rather than backward into the posterior fossa, by reason of the relative thickness of the bone in these two directions. According to Jacod the cranial nerves are involved within the skull at a point which he speaks of as the 'carrefour pétrosphénoïdal—that is, the situation at which the antero-internal end of the petrous temporal comes into contact with the great wing of the sphenoid. Here the first six cranial nerves are placed in close apposition to one another, and may be involved by a comparatively small growth. M. Jacod states that in the peritubular sarcoma the first six cranial nerves are invariably attacked sooner or later, while those which leave the skull in the posterior fossa are never involved. Paralysis of the sympathetic has been noted by him, and he holds that invasion of this nerve occurs in the cavernous sinus.

In my own experience, and in the experience of other authors whose work I have quoted, the nervous signs are by no means as regular as this, and the invasion is definitely not confined to the first six cranial nerves. Neither is invasion of the 2nd, 3rd, and 4th nerves by any means frequent. One only of my cases showed involvement of these nerves, and the proportion in the

'epitheliomata' reported by Woltman is strikingly low. An isolated 6th-nerve paralysis produced by intracranial invasion arouses no surprise, but a prolonged and isolated involvement of one or two parts only of the 5th nerve suggests much more strongly an extracranial invasion. One would have expected also that a growth so accurately localized at the 'carrefour pétrosphénoïdal' would, in a proportion of cases, give rise to a thrombosis of the cavernous sinus. There is no mention of this event in any of the articles which I have consulted. The invasion of the sympathetic has been held by some authors to be due to glandular metastases, but I think this theory is untenable, as in one of my own cases there was a sympathetic paralysis although no enlarged glands could be felt in the neck. Most probably, I think, it occurs before the sympathetic comes into relation with the cavernous sinus.

In the article by M. Jacod referred to above, two cases are quoted. I believe the same author made a previous communication on the subject in the year 1920, but this I have been unable to trace.

Reverchon[6] reports fully two cases, giving in one of them a record of the post-mortem examination. At the post-mortem of this case a mass of growth was found covered by intact dura mater in the middle fossa behind the great wing of the sphenoid. Growth had invaded the sphenoidal sinus, and outside the skull it occupied "all the recesses of the surrounding regions". The second case is mainly remarkable for the fact that in the early stages there was a transient attack of herpes zoster in the distribution of the 5th nerve on the side of the tumour. In both these cases Reverchon refers to 'phénomènes d'hypertension' which in the first case were relieved by lumbar puncture and in the second by a subtemporal decompression. Treatment by radium met with some success in both cases; indeed, it is recorded of the second that after a year of treatment the weight of the patient had increased by 5 kilo. M. Monod, of the Radium Institute, is quoted as giving the following opinion as regards treatment by radium: "Il est vain d'essayer dans un cas semblable, d'atteindre tous les prolongements par la radiopuncture. Si le traitement par le radium est indiqué il faut avoir recours à des appareils moulés recouvrant complètement la région à irradier." An apparatus covering practically the whole of one side of the face and temporal regions would thus be necessary, and a sufficient quantity of radium would be required to irradiate adequately the whole of this region in one application. I speak without experience of this form of irradiation, but I believe that in the present state of our knowledge of the technique of irradiation the difficulties would be very considerable both in attaining a sufficient concentration at a situation so far from the surface and in avoiding damage to normal structures.

The opinion of M. Monod in this matter is, of course, entitled to the very greatest respect. Nevertheless, provided that the presence of the tumour is recognized at a reasonably early period and that the region concerned is adequately exposed by a well-planned operation, I think that there are good prospects of success in treatment by implantation of radium emanation. Naturally success in treatment by implantation depends on an accurate knowledge on the part of the surgeon of the directions in which the tumour ordinarily spreads, and I hope that my article will be found of use in attaining this knowledge.

SUMMARY.

1. An account is given of the clinical characteristics of a tumour usually described as an endothelioma of the nasopharynx. The variability of the clinical picture presented by these tumours is emphasized.

2. It is suggested that tumours of this type are by no means uncommon, and that many of them pass unrecognized because of the absence of obvious symptoms pointing to a nasopharyngeal origin, and because the presence of a tumour is only recognized when the condition is sufficiently far advanced to render the primary origin of the growth a matter of conjecture, and of purely theoretical interest.

3. It is emphasized that there is some evidence to show that endotheliomata in this region react favourably to radium except when it is applied only to the outlying parts of an extensive tumour, and it is suggested that good results might be obtained by a combination of adequate exposure of the growth with irradiation by means of seedling tubes of radium implanted in the directions in which the tumour is known to extend.

I am much indebted to Professor C. C. Choyce for allowing me an unstinted use of the clinical and laboratory facilities of the Surgical Unit; to members of the surgical staff of University College Hospital for permission to make use of their case records, and in particular to Mr. Julian Taylor for his helpful criticism and advice.

REFERENCES.

[1] TROTTER, W., "On Certain Clinically Obscure Tumours Infiltrating the Naso-pharyngeal Wall", *Trans. Med. Soc. London*, 1911, xxxiv.

[2] TURNER, ALDREN, *Jour. Anat. and Physiol.*, xxiii, 1889.

[3] BASTIAN, *Paralysis, Cerebral, Bulbar, and Spinal*, 446.

[4] RICH, A. R., "The Innervation of the Tensor Veli Palatini and Levator Veli Palatini", *Johns Hopkins Hosp. Bull.*, 1920, xxxi.

[5] NEW, G. B., "Syndrome of Malignant Tumours of the Nasopharynx", *Jour. Amer. Med. Assoc.*, 1922, lxxix.

[6] REVERCHON, L., "Deux Cas de Tumeur du Rhinopharynx à Point de Départ péritubaire", etc., *Rev. de Laryngol., d'Otol. et de Rhinol.*, 1923, xliv.

[7] ELOESSER, L., and READ, J. M., "Nasopharyngeal Endothelioma", *Surg. Gynecol. and Obst.*, 1921, xxxii.

[8] FINDER, G., "Zwei Fälle von Endotheliom des harten Gaumes", *Wien. med. Woch.*, 1921, lxxi.

[9] HARMER and GLAS, "Die Malignen Tumoren der inneren Nase", *Deut. Zeits. f. Chir.*, 1907, lxxxix.

[10] POLLAK, E., "Ueber die endothelialen Geschwülste der Nase und ihrer Nebenhöhlen', *Arch. f. Laryng. und Rhin.*, 1911, xxv.

[11] WOLTMAN, H. W., "Malignant Tumours of the Nasopharynx with Involvement of the Nervous System", *Arch. of Neurol. and Psychiat.*, 1922, viii.

[12] CROW, S. J., and BAYLOR, J. W., "Benign and Malignant Growths of the Nasopharynx and their Treatment with Radium", *Arch. of Surg.*, 1923, vi.

[13] STÄHLI, "Ueber Augen Muskellähmung als ein initiales Symptom von malignem Nasenrachen Tumor", *Klin. Monats. f. Augenh.*, 1920, lxv.

[14] JACOD, M., "Sur la Propagation intracranienne des Sarcomes de la Trompe d'Eustache; Syndrome du Carrefour pétrosphénoïdal", *Rev. neurol.*, 1921, xxviii. (Also quoted by Reverchon.)

TWO CASES OF RIEDEL'S CHRONIC THYROIDITIS.

By HELEN F. WINGATE,

EXTRA SURGEON TO OUT-PATIENT DEPARTMENT, GLASGOW ROYAL INFIRMARY.

In view of the rarity of this condition it has been thought worth while to add two more to the number of published cases. A. F. Bernard Shaw and R. P. Smith[1] reported on six cases, and gave a review of the literature, in the British Journal of Surgery in 1925. Both the following cases bear a close resemblance to the earlier ones of their series, or to what used to be termed Hashimoto's struma lymphomatosa. As has so often happened, the diagnosis was only made microscopically, and in the first case with considerable difficulty.

Case 1.—The patient, a woman of 46, was transferred from the medical wards with a marked enlargement of the thyroid gland involving mainly the right lobe, which was smooth and very firm in consistence. She gave a history of a swelling in the neck of six years' duration, with a fairly rapid increase in

Fig. 158.—*Case* 1. Showing two lymph follicles and acini amongst the granuloma. (× 100.)

size during the last three months. Her complaint was of slight dyspnœa, occasional hoarseness, and occasional palpitation, especially on exertion. She stated that she was easily tired, but her general health seemed good, although she showed signs of slight thyroid deficiency. There was no evidence of tuberculosis, and the Wassermann reaction was negative. The pulse-rate

was 80 per minute. The condition was thought to be a parenchymatous goitre, but the question of malignancy was considered owing to the unusual hardness of the gland.

OPERATION.—The right lobe and isthmus of the thyroid were removed with some difficulty owing to adhesions to the surrounding structures. There was some thickening of the capsule. The cut surface of the gland was finely lobulated, opaque, and solid looking, and seemed entirely devoid of colloid.

The patient was seen recently, two and a half years after operation. A definite thyroid deficiency had been counteracted by the administration of thyroid extract, and she is now feeling very well. There has been no recurrence of the swelling, and the left lobe of the thyroid has gradually decreased in size.

HISTOLOGY. — The capsule and interlobular septa are all infiltrated with mononuclear cells. The vessels are scanty and of normal thickness. The appearances in different places vary considerably. In some parts, presumably where the disease is more advanced, hyaline stroma forms a coarse reticulum enclosing comparatively few cells and no acini. Other parts show a very cellular granuloma, with a minimal amount of stroma and enormous numbers of plasma cells, lymphocytes, and a few fibroblasts. In some of these areas acini are present in considerable numbers. Some contain a small amount of colloid. Where acini are unrecognizable high-power examination sometimes reveals the remains of epithelial cells, many invaded by leucocytes or plasma cells which are apparently acting as phagocytes.

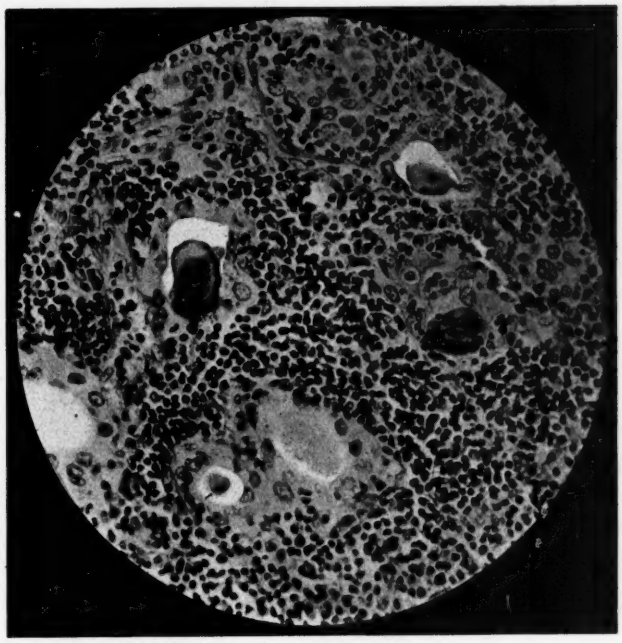


FIG. 159.—*Case* 1. An acinus is seen to the right of the centre containing a foreign-body giant cell and phagocytes. To the left is an acinus containing colloid which is undergoing phagocytosis. (× 200.)

The most striking feature in all the sections examined is the presence of very definite and well-formed lymph follicles (*Fig.* 158). These vary in size, and are round or oval in shape. Each germ centre is surrounded by a definite zone of lymphocytes which varies in thickness and is limited by a delicate stroma containing capillaries. Among the cells of the follicle a considerable amount of irregular hyaline material is seen. The majority of the cells are large and round with vesicular nuclei, but lymphocytes and fibroblasts are also present. These follicles are to be found throughout the sections.

Another notable feature is the number of giant cells to be seen with a low power (*Fig.* 159). They are unevenly distributed and are not associated with the lymph follicles. With a higher power many of these are found to be masses of colloid invaded by phagocytic cells which are at times arranged in a circle. These masses are usually to be seen within acini. The most striking of the real giant cells are strongly suggestive of tubercle, and are, in our opinion, foreign-body giant cells. They are commonly embedded in degenerating acini, their cytoplasm being readily distinguishable from that of the epithelium by slight differences in texture and staining. The nuclei are smaller than those of the epithelium and they are arranged in characteristic groups. As already mentioned, the acini may also show invasion by plasma cells. In places these giant cells are actually within acini, but we could find no evidence that they were derived from the epithelium, although a multinucleate condition of the epithelial cells may occasionally be seen.

Case 2.—The patient, a woman of 34, gave a history of a swelling in the neck of five years' duration, associated for the last year with severe attacks of dyspnœa which had caused her to seek advice. The right lobe and isthmus of the thyroid were enlarged, the swelling being smooth and apparently cystic. She showed signs of slight hyperthyroidism, but the pulse-rate was only 86 per minute. The Wassermann reaction was negative.

Operation.—Although but slightly enlarged, the right lobe of the thyroid was removed with great difficulty owing to adhesions to surrounding structures, particularly the trachea. The cut surface of the isthmus was normal in appearance, while that of the right lobe was of an opaque white colour broken in places by vascular strands and studded with yellowish areas of colloid.

Histology.—There is much thickening and cellular infiltration of the capsule and interlobular septa and some sclerosis of the blood-vessels. The typical granuloma with large numbers of lymph follicles and very marked fibrosis composes most of the gland, but, in striking contrast to the first case, there are areas of comparatively normal acini. Some contain colloid and others show absorption of colloid and definite signs of activity. Here also masses of colloid can be seen invaded by phagocytic cells, but the foreign-body giant cells are very scanty.

Both these cases appear to be examples of Riedel's chronic thyroiditis. Histologically the lesion in the first is at an earlier stage. Although the gland parenchyma is much more extensively destroyed, the granuloma is more cellular and there is less fibrosis than in the second case.

While differing in our interpretation of the giant cells, we agree with Bernard Shaw and Smith that the lymph follicles represent hyperplasia of the minute lymphatic nodes to be found in so many organs, and that the true nature of the condition is that of a non-tuberculous granuloma.

I have to thank Mr. Kay, Mr. Milne McIntyre, and Professor John H. Teacher for permission to publish, and Professor Teacher for the photographs and for much helpful criticism.

REFERENCE.

[1] Shaw, A. F. Bernard, and Smith, R. P., *Brit. Jour. Surg.*, 1925, July, 93.

REPORT OF A CASE OF ANEURYSM OF THE SPLENIC ARTERY:

WITH REFERENCES TO 58 CASES COLLECTED BY THE AUTHORS.

By WILLIAM ANDERSON,

ASSISTANT SURGEON TO THE ABERDEEN ROYAL INFIRMARY;

AND JOHN GRAY,

ASSISTANT IN THE PATHOLOGICAL DEPARTMENT IN THE UNIVERSITY OF ABERDEEN.

Aneurysms of the branches of the abdominal aorta have been looked upon as pathological rarities, but there have been so many instances recorded recently that their recognition as a cause of clinical symptoms becomes of increasing importance. In 1928 Thomson[1] was able to collect 65 cases of aneurysm of the hepatic artery, and Senger[2] 40 cases of aneurysm of the renal artery. We have been able to find in the literature 58 cases of aneurysm of the splenic artery.

CASE REPORT.

Mrs. M., age 49, was admitted on Dec. 17, 1926, at 11 p.m., suffering from agonizing pain in the abdomen of seven hours' duration.

Previous History.—The patient was too ill to give a detailed history, but her husband said that for some years she had suffered from 'indigestion'—pain and discomfort coming on three to three and a half hours after meals, and occasional sickness and vomiting. There was no history of hæmatemesis or of melæna. She was indefinite about her menstrual history—thought the last period was two weeks ago, but stated that for some time there had been irregularity.

Present Illness.—About 4 p.m. on the day of admission the pain came on suddenly, and she collapsed but did not become unconscious. She vomited a little, but did not notice the nature of the vomitus. The agonizing pain continued till the time of her admission.

On Examination.—Patient was anæmic and collapsed; temperature 98°, pulse 126, respirations 24. The abdomen was somewhat distended, tender all over, but with no muscular rigidity. Liver dullness was present, and ? dullness in both flanks. Pelvic examination was negative. No gross lesion of heart and lungs was made out. The provisional diagnosis was intraperitoneal hæmorrhage; ? ectopic gestation.

Operation (W. A.).—A right paracentral incision was made. A large quantity of fluid blood escaped from the peritoneal cavity, and a few soft blood-clots were removed from the pelvis. Examination of the pelvic organs showed the uterus to be slightly larger than normal, and the extremity of

the left Fallopian tube to be somewhat patulous. A rapid examination of the rest of the abdomen did not reveal any gross abnormality. Thinking I was dealing with a left tubal abortion I quickly removed the outer half of the tube and closed the abdomen. A pint of normal saline was given intravenously.

Convalescence was somewhat stormy for five days, the temperature being as high as 102° the day following operation, but thereafter the patient did well and was almost ready to leave hospital. On Jan. 4 she was reading in bed when she asked for a bed-pan ; before it could be brought she collapsed and died within twenty minutes. The symptoms suggested a pulmonary embolism.

Professor Low, Anatomy Department, Aberdeen University, kindly undertook the examination of the Fallopian tube, but careful serial section study failed to reveal any evidence of pregnancy.

Post-mortem Examination (J. G.).—The body was very pale. The greater sac of the peritoneal cavity contained a large amount of blood-clot and fluid blood of evidently recent origin. The lesser sac also was distended with blood. Some of this was fluid blood or recent clot similar to that in the greater sac, but there was a thick, shaggy layer of older clot adherent to the wall of the lesser sac throughout. At one point on the posterior wall there was an irregularly rounded crater-like depression in this layer of older clot, and that point was found to lie immediately over a saccular aneurysm of the splenic artery. A small chink-like opening in the wall of the aneurysm communicated with the lesser sac at this site. It was evident that two hæmorrhages had occurred from the aneurysm at different dates.

The first hæmorrhage, which, from the history, had evidently immediately preceded the operation, marked the time of the initial rupture of the aneurysm into the lesser sac. Hæmorrhage had at that time extended from the lesser sac through the foramen of Winslow into the greater sac, and accounted for the blood found there at operation. Such of the blood in the greater sac as had not been removed at operation was apparently absorbed subsequently. Much of the blood in the lesser sac had, however, remained, and formed the older, more adherent clot just described there. This clotting temporarily closed the opening in the aneurysmal wall.

The second and fatal hæmorrhage, occurring eighteen days after, burst through the same opening in the aneurysm into the lesser sac, ploughing up the older clot on the wall of the sac and passing by way of the foramen of Winslow to the greater sac.

The aneurysm itself was a 'false' aneurysm, a rather rough-walled sac forming a more or less rounded cavity about the size of a cherry lying in the substance of the pancreas. It opened from the main splenic artery by a narrow gap about half an inch long on the antero-inferior aspect of that artery at a point just outside the hilus of the spleen.

To the naked eye there was no atheroma of the splenic artery or of the main trunk of the cœliac axis. There was a small amount of blood-clot around the pancreas. The uterus and the uterine appendages on the right side were healthy. Beyond marked anæmia there were no important alterations in other organs.

MICROSCOPIC EXAMINATION.—

Splenic Artery Proximal to the Site of the Aneurysm.—The outer half of the media appeared to be almost normal. The inner half, however, showed extensive partial necrosis of the muscle fibres, the muscle nuclei being broken up or greatly distorted where they had not actually disappeared. A rather surprising feature was the practical absence of accompanying cellular infiltration, only a few small round cells being visible in the patches. There was no vascularization or evidence of increase of connective tissue. The elastic tissue of the degenerated part of the media was granular and was demonstrated with difficulty. Some fibrin was present. The intima was not thickened, and nothing abnormal was noted in the adventitia save perhaps a slightly increased cellularity (small round cells).

Splenic Artery at the Opening into the Aneurysm.—Sections were made showing the ruptured splenic artery opening into the aneurysmal sac. The wall of the artery here showed more marked disease than that just described in the wall nearer the commencement of the artery, but the changes appeared to be of the same type.

The only coat affected around the whole circumference was the media, and here, as before, the more marked alterations were in its inner half. All round, the inner half showed extensive necrosis of the muscle fibres with karyorrhexis of the muscle nuclei which survived. There was more round-celled infiltration in this zone than in the corresponding zone in the previous sections, but it was not very marked. There was no definite vascularization or increase of connective tissue. The outer half of the media showed an extensive patchy loss of individual fibres and groups of fibres, granular fibrinous débris alone surviving in such patches.

The intima was not thickened all round, and in particular it was not altered at the point of connection with the aneurysm (on the side investigated). It showed, however, considerable cellular thickening in part of the circumference. In this part, the internal elastic lamina, which had been running close to the endothelium, suddenly passed outwards for a short distance, to disappear abruptly. The thickened intima here was composed of cellular connective tissue, the cells being mainly fibroblasts with some small round cells. A few capillaries were visible.

There was no fatty change in either intima or media save for a very fine dusting of the internal elastic lamina. There was extensive fibrosis of the surrounding pancreatic tissue, but no cellular infiltration of the adventitia.

The opening into the aneurysm was next studied. The vessel wall approaching this point showed an unthickened intima but a very extensive necrosis of the media, leaving only a narrow surviving band of relatively healthy muscle in its outer part. Opposite the aneurysmal opening this wisp of media stopped abruptly, a small hæmorrhage marking the site of its termination in some sections. Shortly afterwards the internal elastic lamina and endothelium stopped also, and finally the adventitia with its thin layer of elastic tissue disappeared. Beyond this no semblance of arterial wall persisted. The aneurysmal wall was formed of concentric layers of fibrosed pancreas, with an inner lining of adherent thrombus.

In certain other sections cutting through the opening of the artery into the aneurysm at a different point, the external and internal elastic laminæ both persisted, one on either side of the degenerated media, right up to where the aneurysm commenced; the media then thinned out and stopped, and as it did so the laminæ approached one another, almost joining where the media stopped. Here the internal elastic lamina stopped abruptly, but the external elastic lamina continued as a few faint strands in the adventitial tissue for a little into the wall of the aneurysm (*Fig.* 160).

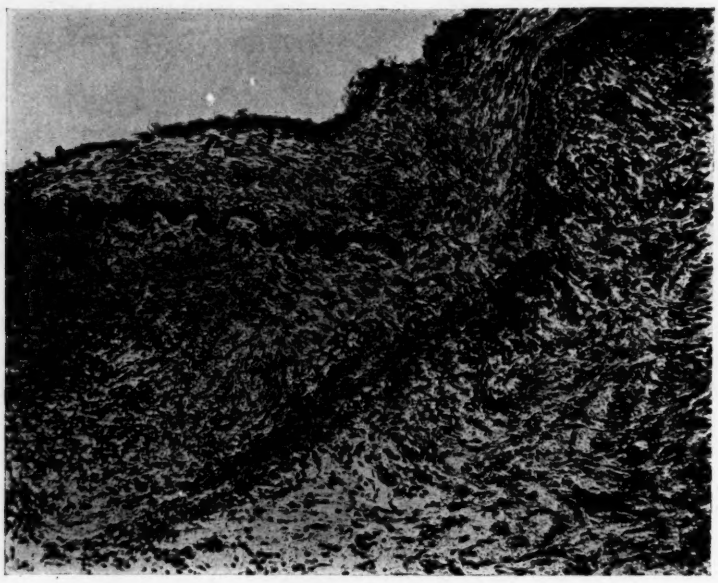


FIG. 160.—Opening into aneurysm. Wall of splenic artery to left, internal and external elastic laminæ converging from left to right, forming a triangle.

Discussion of Microscopic Appearances.—It seems clear that the medial degeneration and necrosis were the primary and chief factors leading to the formation of the aneurysm. The thickened intima found in parts is apparently secondary, representing an attempt to strengthen the weakened wall, and it may be noted that rupture occurred at a point where this thickening had not taken place. The process seems to have been of fairly recent origin, but the paucity of cellular infiltration is peculiar.

There is evidently no relation to atheroma or other generalized arterial disease. The appearances do not suggest syphilis. They do, however, suggest a subacute infection.

COMMENTS ON CASES.

This unfortunate experience induced us to look up the literature, and we were struck by our ignorance of the number of recorded cases. In the accompanying table (*see* p. 272) the chief features of 58 cases are summarized.

Etiology.—A study of these cases does not show any outstanding causative factor. Septic emboli, arteriosclerosis, and syphilis have been suggested, but in a number of instances all of these can be definitely excluded. A Wassermann reaction was not done in our case, but the family history did not suggest syphilis, nor did post-mortem examination reveal any stigmata of this disease. No history of a previous septic focus could be obtained, nor was one found post mortem. Direct or indirect trauma has been suggested as a cause in a few cases. There was no history of this in the present case, although the patient had lived a strenuous life as the wife of a small farmer.

Symptoms.—A history of discomfort in the left upper abdomen often closely simulating a gastric or duodenal ulcer was given in a number of the cases. In others a definite enlargement of the spleen or tumour mass in the left upper abdomen could be made out, while in still others the patient gave no history to suggest the presence of an abdominal lesion. When perforation occurs the picture is that of internal hæmorrhage, and in the female ectopic gestation has been the pre-operative diagnosis in a number of instances. When rupture of the aneurysm occurs in the late months of pregnancy the difficulties of diagnosis are very great indeed, and a concealed intra-uterine hæmorrhage is likely to be suspected.

Treatment.—Surgery offers the only hope of cure, and we believe that a wider appreciation of the possibility of the lesion will, as in other conditions, lead to earlier diagnosis and better results.

REFERENCES.

[1] THOMSON, *Johns Hopkins Hosp. Bull.*, 1928, March.
[2] SENGER, *Arch. of Pathol.*, 1928, Feb.

Table showing the references to 58 cases follows

TABLE SHOWING THE REFERENCES TO THE 58 CASES COLLECTED BY THE AUTHORS.

No.	Author	Sex Age	Symptoms	Aneurysm	Treatment (Operation)	Results	References
1	Ahrens, M.	M. 26	Pressure and pain in stomach region, vomiting of blood; swelling of spleen; small pulsating tumours palpated	Size of a fist; ruptured into peritoneum	Patient died before treatment could be begun	Sudden death with signs of internal hæmorrhage	*Inaug. Diss.*, Griefswald, 1892
2	Ayer, J. B.	F. 44	Hæmatemesis; severe abdominal pain	Rupture into stomach not stated	Morphine only mentioned	Sudden death	*Boston Med. and Surg. Jour.*, 1883, cviii, 148.
3	Barlow	M. 61	No symptoms	Two aneurysms: 1 size of filbert nut, and 1 size of pea, about 1 in. apart	Not stated	Death	*Trans. Pathol. Soc. Lond.*, 1899, i, 57
4	Baumgartner, E. A.	F. 48	Sudden abdominal pain; vomiting blood, and blood in stools; indigestion for some time	9 cm. in diameter, not ruptured	None	Death	*Surg. Gynecol. and Obst.*, 1924, xxxix, 462
5	Beaussenat	M. 54	Ulcerations on legs; œdema of left leg; stomach dilated; constipation	Dilatation, 35 cm. long, sac 10 cm. in length, circumference of hen's egg	Local for ulcers; incisions for œdema	Death	*Anat. Ges.*, Paris, 1892
6	Beaussier	F. 60	—	One size of small walnut, others smaller	—	—	*Jour. of Med.-Chir. Pharm.*, 1770, xxxii, 157
7	Berry, J. A.	F. 60	Those of carcinoma of stomach, which was present	Main artery and branches; 5 aneurysms, largest 1 in. in diameter	None	Death (from cancer of gall-bladder)	*Lancet*, 1927, i, 490
8	Binder	M. 47	Severe pain in region of spleen; later colicky pain; spleen enlarged	Size of walnut, ruptured into peritoneum	None	Death day before operation	*Bruns' Beitr. z. klin. Chir.*, 1918, iii, 205
9	Corson, E. N.	The original article was not seen					*Med. and Surg. Reporter*, 1869, xx, 351
10	Crisp	M.	The original article was not seen			Death	Quoted by Zahn, 1847
11	Davidson, A.	—	—	One size of bean, 1 size of pea, and others smaller still in branches of artery	—	—	*Liverpool Med.-Chir. Jour.*, 1884, iv, 210
12	Davis, B. F.	M. 67	Sudden appearance of	One	Removed	Recovery	*Jour. Amer. Med.*

11	Davidson, A.	—	—	One size of bean, 1 size of pea, and others smaller still in branches of artery	—	—	*Liverpool Med.-Chir. Jour.*, 1884, iv, 210
12	Davis, B. F.	M. 67	Sudden appearance of tumour to left of umbilicus	One	Removed	Recovery	*Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 200; also *Med. Press*, 1925, N. S. cxix, 115
13	Faehrmann and Ugrumow	M. 50	Splenomegalia	8 cm. in circumference, 3 cm. from site of ligature	Splenectomy	Death	*Arch. f. klin. Chir.*, 1925, cxxxvii, 193
14	Fitzwilliams, D. C. L.	F. 27	Sudden intense pain in pit of stomach; collapse, shock	Three aneurysms, largest (ruptured, size of walnut) in hilus	Exploratory laparotomy	Death	*Brit. Med. Jour.*, 1924, ii, 803
15	Garland	M. 52	Dyspnœa on exertion; œdema	Ruptured into peritoneum. Sac about 3 cm. in diameter	Ruptured. Caused by abdominal paracentesis	Death	*Boston Med. and Surg. Jour.*, 1921, clxxxiv, 385
16	Goodheart, J. F.	F. 49	Abdominal distension; headache, vomiting, and diarrhœa; thirst; loud blowing murmur on left side; hæmorrhage per rectum	Size of hen's egg	None beyond tapping	Death	*Trans. Pathol. Soc. Lond.*, 1889, xl, 67
17	Goullioud	F. 59	Tumour in left hypochondriac region for 4 or 5 years	Size of an orange. Contained large clot	Operation: removal of aneurysm	Recovery. Well after 12 years	*Bull. et Mém Soc. nat. de Chir.*, 1928, liv, 402
18	Harnett	—	—	Traumatic aneurysm	—	—	*Ind. Med. Gaz.*, 1922, 457, lvii
19	Heppner	F. 58	Spleen greatly enlarged; pulsating tumour palpable and at times visible; occasional vomiting and diarrhœa; abdominal pain off and on for some years previously	Three aneurysms, 7·5, 6·0, 5·0 cm., with smaller ones attached. Ruptured into peritoneum	—	Sudden death from internal hæmorrhage	*St. Petersb. med. Zeits.* 1872, N. S. iii, 220
20	Hoegler	F. 61	Swelling in left hypochondrium with bruit; girdle pains	One aneurysm (diagnosed during life as cancer of pancreas)	—	Death (cancer of pancreas also found)	Quoted by Garland, *Wien. Arch. f. int. Med.*, 1920, Aug., 543
21	Hunt	M. 70	Cerebral hæmorrhage	No details given. Rupture into substance of pancreas	None	Death	*Amer. Jour. Med. Sci.*, clxxvi, No. II, 195
22	Jourdan	No details given. The whole communication is as follows: "M. Jourdan showed a piece of aneurysm of the splenic artery burrowed into the head of the pancreas, and claimed that death was due to rupture into the peritoneum"					*Anat. Ges.*, Paris, 1881, iii, 76

Continued on next page

TABLE SHOWING THE REFERENCES TO THE 58 CASES COLLECTED BY THE AUTHORS—*continued*.

No.	Author	Sex Age	Symptoms	Aneurysm	Treatment (Operation)	Results	References
23	Leudet	F. 70	—	Two tumours: 1 size of pea; 1 size of filbert nut	—	—	*Gaz. des Hôp.*, 1852, xxv, 583
24	Lindbom	M. 29	Severe abdominal pain. Swelling and pain in right shoulder	2·5 cm. long, 1·5 cm. broad	Drugs only	Died suddenly 8 days after admission	*Zit. Thereb. Pathol. der Kreislauforgan Lubarsch, Ostertag*, 1915
25	Lundwall and Gödl	F. 23	Internal hæmorrhage. in 9th month of pregnancy	Ruptured. 2 cm. in diameter 3 cm. from origin	Supravaginal hysterectomy	Death	*Arch. f. Gynäkol.*, 1923, cxviii, 177. Abst.: *Jour. Amer. Med. Assoc.*, 1923, lxxx, 1348
26	Marshall	F. 27	—	Traumatic (revolver)	—	—	*Brit. Jour. Surg.*, 1921–2, ix, 570
27	Mayer	F. 29	Severe lower abdominal pain in 1st stage labour	Size of hazel-nut. In main artery. Ruptured just before division. Pancreatic hæmatoma	—	Died 1½ hours after delivery	*Zentralb. f. Gynäkol.*, 1928, lii, 754
28	Mayet	F. 59	Abdominal pain for 2 months before; vomiting after meals; loss of weight; hæmatemesis	Size of small nut. Ruptured into stomach	Alkaline waters	Death	*Ann. Soc. méd. de Lyon*, 1879, xxxi, 327
29	Monro	M. 23	Breathlessness on exertion; pain in chest and legs; swelling of feet.	Spherical, about size of tangerine orange. Filled with clot	Injections of anti-streptococcus serum. No apparent benefit, but seemed to cause increase in urine	Death	*Trans. Glasgow Pathol. and Clin. Soc.*, 1905–6, xi, 92; also *Glasgow Med. Jour.*, 1907, lxvii, 309
30	Mulley	F. 23	Anæmia; constipation; vertigo and faintness	Two aneurysms: 1 size of small apple, and 1 size of nut	Splenectomy	Complete healing	*Beitr. z. klin. Chir.*, 1918, cxi, 205
31	Näher	F. 54	Feeling of fullness in upper abdomen for 3 months; tumour in left upper abdomen which slowly increased in size, noted for more than 1 year before; no great pain, no vomiting, no œdema; urine normal	Not stated	Operation: Jan. 12, 1925. Removal of spleen with tumour	Complete recovery without complications. Discharged 14 days. In good health 17 months later	*Deut. Zeits. f. Chir.*, 1926, cxcviii, 118
32	Osler	M. 30	Tumour in left hypochondrium	Size of coconut	—	Death	*Montreal Gen. Hosp.*

			noted for more than 1 year before ; no great pain, no vomiting, no œdema ; urine normal			later	
32	Osler	M. 30	Tumour in left hypochondrium	Size of coconut	—	Death	*Montreal Gen. Hosp. Rep.*, 1880, i, 266
33	Parker	M. (Age not given)	Rectal hæmorrhage ; slight dyspepsia ; pain in back	Size of small orange	None	Death 1½ hours after admission to hospital	*Edin. Med. and Surg. Jour.*, 1844, lxii, 132 ; also *Dublin Quart. Jour. Med. Sci.*, 1844, xxvi, 124
34	Remmelts	F. 38	Sudden attack of severe abdominal pain at end of pregnancy	2 cm. long	It was intended to operate, but patient died during preparation for laparotomy	Death	*Nederl. Tijds. v. Verlosk. en Gynæc.*, 1928, xxxiii, 41 ; also trans. : *Zentralb. f. Gynäkol.*, 1928, lii, 167
35	(*a*) Reynolds			The original article was not seen			*S. Afric. Med. Record*, 1906, iv, 267
	(*b*) van Rooy			The same case, original not seen			*Nederl. Maandschr. v. Geneesk.*, 1927, xiv, 507 (in Dutch)
36	Rolleston	F. 37	Not stated. Post-mortem description	Branch. Size of a cherry	—	Death	*Trans. Pathol. Soc. Lond.*, 1898–9, l, 55
37	Saenger	F. 40	Internal hæmorrhage	Size of hazel-nut 6 cm. from hilum	Operation	Death	*Zentralb. f. Gynäkol.*, 1926, i, 1324
38	Santesson			The original article was not seen			*Förth Svens. Läk-Sallsk. Sammank.*, 1848–9, Stockholm, 1849
39	Schroeder	F. 32	Hæmolytic jaundice and splenomegalia	Two aneurysms. 1 9 cm. from hilum ; 1 at hilum, size of hazel-nut	Operation for splenectomy abandoned	Death	*Arch. f. klin. Chir.*, 1924, cxxxii, 175
40	Schultze	F. 62	None	Ruptured aneurysm at middle of the pancreas. Found post mortem	—	Death	*Zeigler. Beitrage*, 1905, xxxviii, 374
41	Selten	F. 31	Hemiplegia	Saccular : accidentally found post mortem	None	Death	*Virchow's Arch.*, 1893, cxxxiv, 189
42	Smith, H. B. W.	F. 35	Abdominal pain	Ruptured into peritoneum. Branch at hilum	—	Death. No definite aneurysm found at site of rupture	*Brit. Med. Jour.*, 1911, 83

Continued on next page.

TABLE SHOWING THE REFERENCES TO THE 58 CASES COLLECTED BY THE AUTHORS—*continued.*

No.	Author	Sex Age	Symptoms	Aneurysm	Treatment (Operation)	Result	References
43	Smith, W. R.	F. 38	Diagnosed as ruptured ectopic gestation	'Large tumour' 3 in. long, middle of pancreas. Retro-peritoneal rupture	Exploratory laparotomy	Death	*Jour. Amer. Med. Assoc.*, 1923, lxxx, 1692
44	Tarrozzi	F. 45	Post-mortem report. Aneurysm not suspected durng life	Size of filbert nut	—	Died of cancer of stomach	*Ital. Lit. ref. Central. f. Psych.*, 1904, xv, 700
45	Taylor	M. 14	Pains in joints; paroxysms of precordial pain	Large	None	Death 2 days after admission	*Eng. Lit.*, 1915; *Glasgow Med. Jour.*, 1911, lxxv, 249.
46	Teacher	F. 43	Pain in left hypochondriac region, constipation, vomiting	Four aneurysms about ½ in. diameter	None	Death 2 days after admission	
47	Trevor, R. S.	M. 53	Entered hospital for hydrocele: operation performed. Great tendency to hæmorrhages. Diarrhœa and hæmorrhage from bowel two days after	Aneurysm of walnut size made up of 2 pouches. Another aneurysm ½ in. away, size of hen's egg. Splenic vein dilated	—	Death 13 days after operation	*Trans. Pathol. Soc. Lond.*, 1903, liv, 302
48	Turner, F. C.	M. 37	Not stated. Sudden death	Size of an orange; rupture into peritoneum	—	Death	*Trans. Pathol. Soc. Lond.*, 1885, xxxvi, 151
49	Villard and Murard	M. 33	Epigastric swelling and pain	—	Cyst opened and packed	Death	*Arch. gén. de. Chir.*, 1912, viii, 749; also *Lyon méd.*, 1912, cxviii, 830
50	Walz	M. 27	Shivering; joint pains; anæmia; spleen enlarged and painful	—	Anti-rheumatic drug treatment	Sudden death	*Deut. med. Woch.*, 1908, xxxiv, 177
51	Ware, J.	F. 72	Diarrhœa; severe abdominal pain; vomiting; retention of urine; abdomen much swollen	Ten aneurysms from ⅛ in. to ½ in. in size, walls ossified	Opiates for pain only	Death one week after examination	*Boston Med. and Surg. Jour.*, 1856, liv, 297
52	Weigert	F. 49	Not given. Post-mortem report only	1·7 cm. diameter. Branch. Ruptured into splenic vein, multiple sizes	Not stated	Death	*Ges. Abhandlungen, Arch. f. pathol. Anat.*, 1886, civ, 26
53	Wesenberg	F. 32	Collapse and death af-	5 cm. long, 1 to 2 cm.	—	Death during la-	*Zentralb. f. Gynäkol.*,

52	Weigert	F. 49	Not given. Post-mortem report only	1·7 cm. diameter. Branch. Ruptured into splenic vein, multiple sizes	Not stated	Death	*Ges. Abhandlungen, Arch. f. pathol. Anat.*, 1886, civ, 26
53	Wesenberg	F. 32	Collapse and death after birth of a still-born child	5 cm. long, 1 to 2 cm. thick at hilum. Ruptured into peritoneum	—	Death during labour	*Zentralb. f. Gynäkol.*, 1912, xxxvi, 463
54	West, S.	M. 56	Hæmorrhage per anum; vomiting	Branch. Ruptured into stomach. Size not given	Not stated	Death	*Lancet*, 1885, 518
55	Winckler	F. 25	Splenomegalia	Not stated. At hilum	Splenectomy	Death	*Zentralb. f. Chir.*, 1905, xxxii, 257
56	Yolland	F. 27	Collapsed, no diagnosis possible	'Small'	None	Death	*Brit. Med. Jour.*, 1925, i, 600
57	Zahn	M. 44	Hæmatemesis and melæna	Size of nut. Perforated into stomach and transverse colon	—	Death	*Virchow's Arch.*, 1891, cxxiv, 238
58	Zannini			The original article was not seen			*Gaz. med. ital. prov. veneti Padova*, 1880, xxiii, 296

SECONDARY HYDROCEPHALUS AS A FACTOR IN THE DIAGNOSIS AND LOCALIZATION OF INTRACRANIAL TUMOURS; WITH ITS INVESTIGATION AND TREATMENT.*

By CECIL P. G. WAKELEY,
SURGEON TO KING'S COLLEGE HOSPITAL, LONDON;
AND I. M. ALLEN,
REGISTRAR TO NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC, LONDON.

SUMMARY OF CONTENTS.

I.—Introduction
II.—The Circulation of the Cerebrospinal Fluid
III.—The Etiology of Hydrocephalus in Intracranial Tumours
IV.—Personal Cases
V.—Clinical Commentary
VI.—Secondary Hydrocephalus as a Factor in Diagnosis
VII.—Secondary Hydrocephalus as a Factor in Localization
VIII.—The Investigation of Secondary Hydrocephalus and the Results Obtained
IX.—The Treatment of Secondary Hydrocephalus
X.—Summary and Conclusions

I. INTRODUCTION.

Though the symptoms of hydrocephalus secondary to an intracranial tumour are usually regarded as being essentially those of the underlying condition, it frequently happens that no localizing signs whatever are present. In such cases the intracranial tumour itself may remain latent throughout the greater part or whole of the clinical course, and the symptoms be entirely those of acute or subacute hydrocephalus—that is to say, those which are usually regarded as the 'general symptoms' of brain tumour, namely, headache, vomiting, vertigo, and papillœdema. In some cases, however, the vertigo may suggest the presence of a tumour in or adjacent to the posterior fossa of the skull, and result in a presumptive localization of the lesion which may be adduced as evidence in favour of a decompression operation below the tentorium rather than above it; or mental symptoms may appear early in the course of the condition and suggest the presence of a tumour in the frontal lobe rather than one in the base of the brain obstructing the outflow of cerebrospinal fluid from the lateral ventricles. Thus 'false localizing signs'

* From the West End Hospital for Diseases of the Nervous System, London.

may appear and lead to an erroneous localization and to fruitless attempts at exploration and removal. False localizing signs have been recognized as occurring frequently as a result of vascular changes and œdema of the brain in association with intracranial tumours, but hitherto they have not been recognized as a potential source of error in those cases in which a tumour of the brain has produced internal hydrocephalus. Even when they have been regarded as sources of error, it has not always been recognized that the key to a complete diagnosis depended upon the diagnosis of hydrocephalus on the basis of the so-called general symptoms of tumour of the brain; or that often the general symptoms present are the result of secondary hydrocephalus, and the localizing signs present are due to the hydrocephalus and not to the tumour itself. Because of the possibility of error in diagnosis in such cases, it has been thought advisable, using seven typical cases as a text, to consider the influence of secondary hydrocephalus upon the diagnosis and localization of intracranial tumours; to consider the means whereby the presence of hydrocephalus as a complication and the exact localization of the tumour may be determined; and to discuss the influence of such a complication upon the choice of treatment.

II. THE CIRCULATION OF THE CEREBROSPINAL FLUID.

Though it was suggested by Faivre[1] in 1854 that the choroid plexuses were the source of the cerebrospinal fluid, it was not until 1919, when Dandy[2] published the results of his experiments, that it was proved that they were its only source. The way in which it is produced is still in doubt, for by different observers it has been thought to be produced by filtration and by secretion. That the constituents of the blood-plasma are present in it in about the same proportions as in the blood seems to indicate that the process is one of filtration, but the holding back of some substances artificially introduced into the blood-stream suggests that the cells of the choroid plexus have some power of selection over the substances which pass into the cerebrospinal fluid.

Though some of the cerebrospinal fluid may pass into the perivascular spaces, it is probable that most of it is absorbed into the veins. The observations of Weed[3] showed that this absorption of cerebrospinal fluid probably takes place in the subarachnoid space by a process of diffusion, but there is some evidence that absorption of cerebrospinal fluid takes place into the spinal veins as well.

The circulation of the cerebrospinal fluid is a more or less independent one of its own within and without the brain and spinal cord, consisting primarily of a steady tide from its source in the choroid plexuses to the area of absorption in the subarachnoid space and the spinal canal, together with certain superadded waves imparted to it by the vascular pulsations operating within the rigid cranium. The fluid constantly accumulates within the lateral ventricles and circulates in only one direction. From the lateral ventricle it passes through the foramina of Monro into the third ventricle, through the aqueduct of Sylvius into the fourth ventricle, and thence, by way of the foramen of Magendie and the foramina of Luschka, into the subarachnoid spaces about the brain and the spinal cord.

III. THE ETIOLOGY OF HYDROCEPHALUS IN INTRACRANIAL TUMOURS.

Four different mechanisms have been suggested to explain the development of hydrocephalus in the course of intracranial tumours :—

1. Direct mechanical obstruction of the circulation of the cerebrospinal fluid depends upon the accidental localization of the tumour in such a position that it directly impinges upon the channels through which the fluid normally circulates. This effect is produced by tumours blocking one or both foramina of Monro, involving the third ventricle, obstructing the aqueduct of Sylvius, involving the fourth ventricle, or obstructing the foramina of Luschka or that of Magendie. The inevitable result of tumours so placed is the accumulation of cerebrospinal fluid above the level of the obstruction. In these cases hydrocephalus and 'general symptoms' usually appear early, and localizing signs are often absent or appear late as 'false localizing signs'. Such true localizing signs as are present are soon obscured by general symptoms and false localizing signs, so that an error in localization is particularly apt to occur. Since, however, in these cases any treatment adopted must be essentially general, at least in the first place, in that it is determined by the general symptoms of the patient, the initiation of effective treatment must depend upon the prompt recognition of the presence of hydrocephalus. Such a prompt diagnosis is particularly necessary in the case of a tumour or other lesion in the sites already mentioned. It is possible that in such cases the primary mechanical obstruction is only partial and that the vicious circle set up by the gradual development of hydrocephalus disturbs the adjacent brain tissue and makes the obstruction complete, thus hastening the development of general symptoms and at once placing the patient in a dangerous condition. Such is the urgency of the problem presented when secondary hydrocephalus is the result of mechanical obstruction to the circulation of the cerebrospinal fluid. It is even more urgent in those cases in which hydrocephalus is produced indirectly by a tumour situated in other parts of the brain, as, under those conditions, the chances of employing effective local treatment are greater. The mechanism by which the tumour produces secondary hydrocephalus in such cases is at present undecided.

2. By some writers the indirect production of hydrocephalus is believed to be due to pressure upon the great vein of Galen, either directly or indirectly, and the consequent increased production of cerebrospinal fluid by the choroid plexuses in the lateral ventricles. Basing his argument partly upon anatomical and partly upon clinical evidence, Stopford[4] has recently described this mechanism as the only important one in the indirect production of hydrocephalus in cases of intracranial tumour. Sargent[5] has, however, described an anatomical feature of the opening of the great vein of Galen into a lacuna at the posterior end of the free border of the falx cerebri which would make this mechanism unlikely in the absence of direct mechanical obstruction of the vein by pressure against adjacent parts. Stopford, however, overcomes this difficulty by pointing out that hydrocephalus is more common in cases of tumour of the posterior part of the cerebrum and of the posterior fossa, and concludes that this localization supports his suggestion that direct pressure

is produced upon the great vein of Galen either from above or below as a result of the unyielding character of the tentorium cerebelli. Dandy[2] believes that obstruction of the great vein of Galen is a rare cause of secondary hydrocephalus. It is possible that it is a cause in some cases and a contributing factor in many.

3. Actual distortion of the brain-stem by lateral displacement and torsion, and secondary obstruction to the circulation of the cerebrospinal fluid at its most vulnerable point in the aqueduct of Sylvius by pressure of the hindbrain against the unyielding edge of the tentorium cerebelli, has been suggested as a more probable explanation. Russell Brain[6] believes that this is the mechanism operating in the majority of cases of intracranial tumour complicated by hydrocephalus, and cites in support of it the frequency with which distortion of the brain-stem is found at autopsy in cases of secondary hydrocephalus.

4. In addition, it has been suggested that the obstruction to the circulation of the cerebrospinal fluid occurs in the neighbourhood of the foramen magnum. An increase in the intracranial volume, especially above the tentorium, results in the expulsion of cerebrospinal fluid, pressure is exerted upon the contents of the posterior fossa of the skull, the structures in the neighbourhood of the foramen magnum are displaced, and the outlets of the cerebrospinal fluid are obstructed. Thus a vicious circle is set up and internal hydrocephalus rapidly develops.

It is possible that the first three of the factors mentioned may all play some part in individual cases; but, whatever mechanism is chiefly responsible, it is important to recognize that, apart from those cases in which direct mechanical obstruction has occurred, the hydrocephalus is the direct result of increased pressure within the cranium and that, once it has begun to develop, a vicious circle is established which soon results in the development of a condition which endangers the vision if not the life of the patient.

It is equally important to recognize that in those cases in which secondary hydrocephalus has developed indirectly, the order in which the symptoms and signs appear is of paramount importance—true localizing signs first if the part affected is not a silent area, general symptoms of increased intracranial pressure, followed by false localizing signs as a result of the increased intracranial pressure. It is true that the different phases may merge into one another, but it is none the less essential that the sequence of events should be remembered if errors in localization are to be avoided.

IV. PERSONAL CASES.

Case 1.—**Glioma and diffuse gliosis of the pons, with secondary hydrocephalus and symptoms suggestive of a cerebellar lesion.**

H. A., male, age 30 years, was admitted on Sept. 11, 1928, complaining of headache, vomiting, loss of vision, dizziness, and hæmatemesis of six weeks' duration.

History.—The patient was perfectly well until six weeks before admission. The illness began with pain in the back of the neck. There had been severe headache on the top of the head, varying somewhat from time to time. Vomiting had been present daily since the onset and was worse when the headache was severe. Blood appeared in the vomitus on one occasion. The vision was less acute, and

the patient had been disinclined to read on account of the headache. He staggered as he walked. The previous history was negative, but two of the patient's family had had tuberculosis, and one had died of it.

On Examination.—The mental condition was normal. There was some concentric narrowing of the visual fields, more obvious on the left side. There was some blurring of the optic discs, but no obvious swelling could be seen. Nystagmus: slight, irregular jerks were present, more on looking to the left than to the right; there was a long, slow swing on looking to the left. Hearing was diminished in both ears, and bone conduction was more prolonged than air conduction. There was no evidence of active disease in either the right or the left ear. Some words were slurred in speaking. The left corneal reflex was sluggish. There was slight weakness of the lower part of the right side of the face for voluntary movements. The other cranial nerves were normal.

In the upper limbs motor power was poor, tone was decreased equally on the two sides, and the deep reflexes were present and equal. In the lower limbs motor power was poor, tone was decreased more in the left leg than in the right, the knee- and ankle-jerks were more active on the right side than on the left, and the plantar reflexes were doubtful. The abdominal reflexes on the right side were less active than those on the left. The patient walked on a broad base, swayed from side to side, and tended to go towards the left. There was tremor of the left hand and arm on extension. With the finger-nose test there was slight tremor on movement towards an object, more obvious in the left hand than in the right. Rebound was not so good in the left arm as in the right. With the heel-knee test there was no obvious abnormality. The pulse-rate was 70; the blood-pressure was 135 systolic, and 90 diastolic. The other systems were normal.

There was tenderness below the external occipital protuberance on percussion. On X-ray examination the left mastoid cells were not so clear as the right.

The cerebrospinal fluid was normal. The Wassermann reaction in the fluid was positive with 5 minimum hæmolytic dilutions. In the blood the Wassermann reaction was negative.

Subsequent Progress.—Sept. 15, 1928.—The patient was shivering and complaining of the cold frequently, a symptom which had been present since the onset of the illness.

Sept. 18.—There was a feeling of chilliness and dampness in the distribution of the first and second divisions of the left trigeminal nerve. On objective examination there was analgesia in the distribution of the ophthalmic division of the left trigeminal nerve, and impairment of pain sensation in that of the maxillary division.

Oct. 3.—The optic discs were slightly more blurred. Vomiting was not so frequent, but headache was more frequent. There was complete analgesia and loss of tactile sensation in the first and second divisions of the left trigeminal nerve.

Oct. 9.—There was definite nystagmus on looking to the left. The patient walked staggering from side to side with the head bent forward on the chest. There was pain in the back and some difficulty in passing urine.

Oct. 11.—Both optic discs were swollen to 4 dioptres. The retinal veins were distended and there were hæmorrhages in the retina.

Oct. 13.—An operation was performed to decompress the posterior fossa and the supratentorial region immediately above it. Above the tentorium the dura mater was very tense on both sides, more so on the left side. Below the tentorium the dura was not unusually tense. Before the operation, while anæsthesia was being induced, there was some difficulty with breathing which was overcome as soon as the intratracheal tube was inserted. About ten minutes after the operation respiration ceased, but recovered with artificial respiration. Six hours after the operation the patient had not recovered consciousness, and the respiration was more rapid and stertorous. Death occurred suddenly.

Post-mortem Findings.—Nothing abnormal was found in the bones of the skull or the dura. The convolutions of the brain were very greatly flattened, pale,

and anæmic. The pons and the medulla appeared to be much flattened, distinctly broadened, and their surface markings were obliterated as if from pressure from within the brain. On section, the lateral ventricles were much distended and the aqueduct of Sylvius was obstructed at the level of the pons. The third ventricle and infundibulum were also distended.

Microscopically, complete vertical sections through the pons and medulla showed, at about the junction of the two, a large, more or less diffuse glioma, varying much in structure in different parts—in some more cellular, in others more fibrillary. The general type was that of a spongioblastoma multiforme, with spindle-shaped, irregular and some gliomatous giant cells. There was a diffuse 'gliosis' or 'gliomatosis' around the more definite area of tumour on the left side of the pons. Some of the little vessels in the tumour and neighbourhood showed aggregations of small lymphocyte-like cells around them.

Case 2.—**Glioma of the cerebellum and secondary hydrocephalus in a child; obesity; symptoms simulating those of a degenerative condition of the cerebellar and pyramidal tracts.** (*Figs.* 161, 162.)

H. G., female, age 4 years, was admitted on Aug. 29, 1928, with a history of difficulty in walking for eighteen months.

HISTORY.—Up to eighteen months before admission the development of the child had been normal. Then it was noticed that she had some difficulty in walking, which became progressively worse until she was unable to stand. Shortly afterwards a squint appeared and gradually became more obvious. Her previous health had been good, and two other children in the family were healthy.

ON EXAMINATION.—The child was intelligent and well-developed. The skull was abnormally large, but no gross abnormality was found radiologically. The gait was very ataxic, and walking without support was impossible. There was a right external strabismus, but no evidence of diplopia could be elicited. The ocular movements were inco-ordinated, but there was no definite nystagmus. The ocular fundi showed definite optic atrophy. The deep reflexes were exaggerated, and the plantar reflexes were extensor on both sides. There was no obvious interference with cutaneous sensibility. Excepting for the presence of 4 cells per c.mm., the cerebrospinal fluid was normal.

At a subsequent examination the following additional signs were noted. The pupils were equal and moderate in size, reacting sluggishly to light and accommodation. The right corneal reflex was sluggish. The left side of the face was weak for voluntary movements. The upper limbs were normal. Motor power and muscle tone were apparently good in the legs; the knee- and ankle-jerks were exaggerated, those on the right side being more active than those on the left. Co-ordination was good in the arms, but much impaired in the legs. There was some urgency of micturition. The skull was rather large, and its circumference (21¾ in.) approximated to that of the chest.

SUBSEQUENT PROGRESS.—Nov. 4, 1928.—The mental condition was good throughout. There was slight variation from time to time in the signs of a pyramidal lesion, the plantar reflexes being sometimes flexor and sometimes extensor. For a period of one week the patient was very drowsy and took little interest in her surroundings.

Dec. 3.—The right lateral ventricle was punctured through an opening in the skull above and behind the right ear. Eighty c.c. of cerebrospinal fluid were withdrawn in small amounts at a time, and replaced at each stage by air. At the end of the operation the patient vomited, the breathing became stertorous, and coma suddenly supervened. On the withdrawal of a small amount of cerebrospinal fluid the breathing became normal and consciousness was regained. The patient slept a little after the operation and for four hours was apparently normal. She then became cyanosed and died suddenly.

X-ray examination after the injection of air showed that the right ventricle

was very large. No air had entered the left ventricle. The appearance of the skull suggested hydrocephalus.

POST-MORTEM FINDINGS.—The brain was distinctly large for a child of this age, showed great general flattening, and gave the impression of a thick bag full

FIG. 161.—*Case* 2. Glioma of the cerebellum, secondary hydrocephalus, and obesity in a child: showing tumour and dilatation of the infundibulum.

of fluid. The infundibulum was ballooned out into a thin-walled sac, and on section the lateral ventricles were much distended and full of fluid, with thinning of the overlying brain tissue. A very irregular sheet of tumour was found over

the cerebellum and the neighbouring parts of the pons and medulla, and around the seventh and eighth cranial nerves on the right side. The tumour formed a thick collar around the medulla, lay in the foramen magnum, and probably caused compression. The naked-eye appearance of the tumour was suggestive

FIG. 162.—*Case* 2. The same as *Fig.* 161 showing dilatation of the third and lateral ventricles, and collar of tumour above the cerebellum.

of endothelioma. Sections, however, showed a highly cellular tumour, actively malignant—gliomatous with numerous small glial cells and a rich glial network—a fibrillary astrocytoma.

Case 3.—**Glioma of the third ventricle and infundibulum, with secondary hydrocephalus and symptoms suggesting a tumour of the frontal lobe.** (*Figs.* 163, 164.)

M. H., female, age 45 years, was admitted on June 14, 1928, complaining of 'a nervous breakdown', irritability, poor memory, headaches, vomiting, and loss of energy of eight months' duration.

HISTORY.—The patient began to suffer from lack of energy about eight months before admission. For many years she had had headaches on top of the head and in the occipital region. During the previous six months they had been severe and

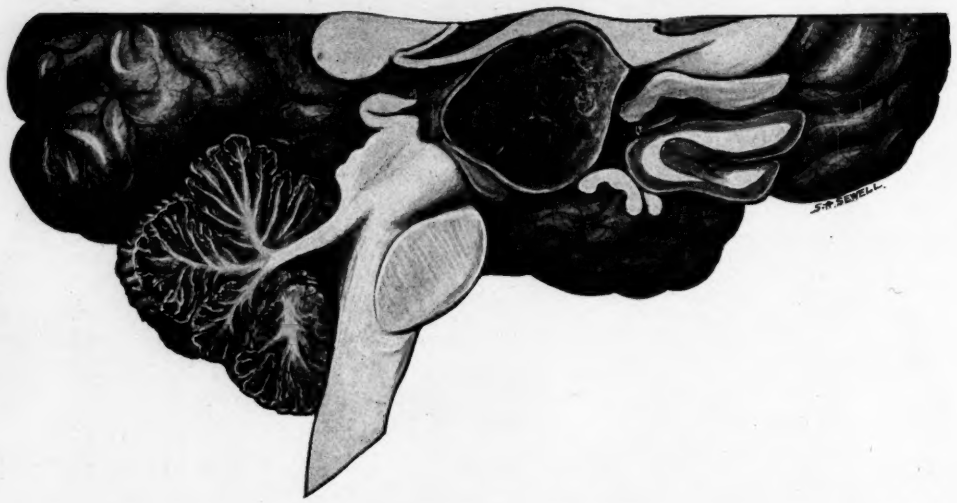


FIG. 163.—*Case* 3. Glioma in the region of the third ventricle and infundibulum with secondary hydrocephalus: sagittal section showing position of the tumour.

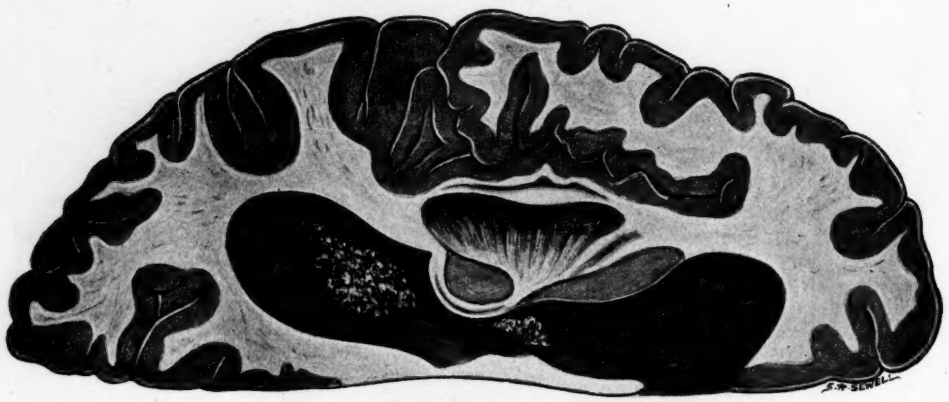


FIG. 164.—*Case* 3. The same as *Fig.* 163, showing dilatation of lateral ventricle.

associated with attacks of vomiting and nausea. During the same time the memory had been getting steadily worse. Although irritable at times, for the most part the patient had been happy and contented, and did not appear to realize the seriousness of her mental deterioration. The previous history was negative. With regard to the family history, the patient's husband was alive and well and she had three children living, one with heart disease.

On Examination.—The mental condition was best described as facile; the patient showed a tendency to joke in everything she said. The memory was very poor, especially for recent events; words spoken a few minutes before could not be recalled. Attention could not be maintained. The right pupil was slightly larger than the left, and both reacted to light and accommodation. The right optic disc was swollen to 2 dioptres, and the left to 4 dioptres with hæmorrhages into the retina. Vision was fairly good. The visual fields could not be measured satisfactorily on account of inattention, but there appeared to be extreme concentric contraction of the fields on both sides. The other cranial nerves were normal.

Sensation was normal. In the upper limbs muscle tone was slightly greater on the right side, and the tendon reflexes were present and equal on the two sides. In the lower limbs muscle tone was slightly greater on the right side, the knee- and ankle-jerks were present and equal, and the plantar reflexes were both flexor. The abdominal reflexes were present and equal. There was tremor of the right hand and arm on extension. The finger-nose test was carried out clumsily, worse on the right side than on the left. There was incontinence of fæces. The blood-pressure was 120 systolic, and 70 diastolic. The other systems were normal.

Subsequent Progress.—A decompression operation was performed in the left fronto-parietal region and the dura opened. No local abnormality was found.

June 27, 1928.—The patient was still comatose twenty-four hours after the operation.

June 28.—The patient was conscious, but unable to speak or swallow. The right arm and leg were spastic, and the right side of the face was paralysed. The temperature was raised, and the pulse- and respiration-rates were increased. Death occurred the following day.

Post-mortem Findings.—The convolutions of the brain showed considerable general flattening due to a moderate degree of hydrocephalic dilatation of the ventricles. In the region of the infundibulum there was a slightly projecting, firm, rounded swelling suggestive of a subjacent tumour. On vertical section this was found to be due to a globular glioma-like tumour about the size of a walnut, centrally placed, and occupying the position of the third ventricle and infundibulum. The tumour was slightly whiter and more spongy than the surrounding brain tissue; and its centre showed some patchy, reddish-brown mottling suggestive of necrosis, with dilated vessels and some small areas of hæmorrhage.

Microscopically, sections showed the tumour to be a glioma of the spongioblastoma multiforme type with considerable variations in the cells, some showing as branching astrocytes, others as spindle-shaped to entirely irregular cells. There were areas of necrosis, and in some parts dilated and congested vessels from some of which hæmorrhage had occurred.

Case 4.—**Endothelioma (meningioma) of the fronto-parietal lobe, with secondary hydrocephalus, glycosuria, and sudden death.** (*Figs.* 165, 166, 167.)

L. P., female, age 27 years, was admitted on Sept. 16, 1927, in a state of coma.

History.—Headache had been present for one year, and gradually increasing drowsiness for three weeks. Mental symptoms had been present for a few days before admission.

On Examination.—The temperature was 97·8° and the pulse-rate 68. The patient was in resistant coma, incontinent of urine, and just reacted to painful stimuli. All the reflexes were increased. There was slight head retraction, and the right side was stiff. The blood-pressure was 124 systolic, and 75 diastolic. The cerebrospinal fluid was not under pressure, was clear, contained 10 lymphocytes per c.mm. and 0·1 per cent of protein. There was a loose cough, but no adventitious sounds were discovered in the chest. The urine was normal.

Subsequent Progress.—Sept. 17, 1927.—The patient could be roused, and spoke about her pain. Papillœdema was present on both sides, on the right side to 3 dioptres and on the left side to 2 dioptres.

Sept. 18.—The coma was increasing, and the limbs showed flaccidity and rigidity at different times.

Sept. 19.—Coma was complete, and the temperature, pulse-rate, and respiration-rate were increasing. The limbs were completely flaccid. The pupils were moderate in size, the left larger than the right, and both were fixed. The abdominal reflexes were absent and plantar reflexes flexor. Severe sensory stimulation caused slight

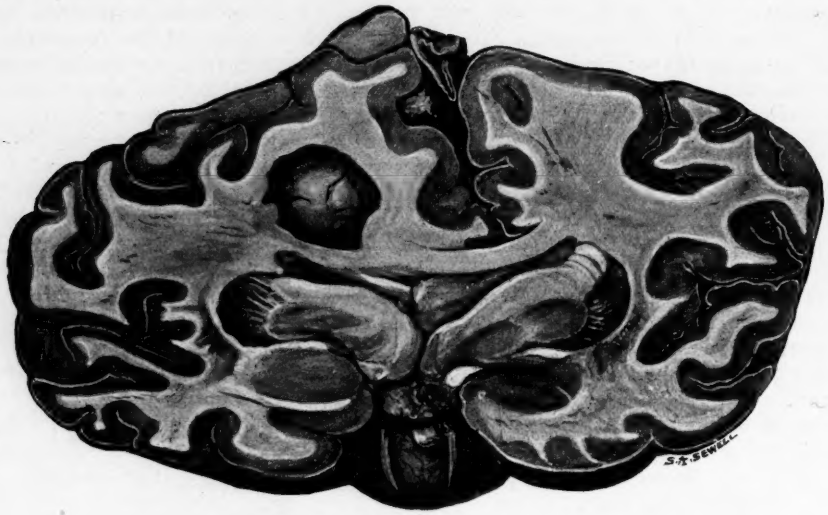


FIG. 165.—*Case* 4. Meningioma of the fronto-parietal lobe with secondary hydrocephalus: showing displacement of structures in the middle line to the opposite side.

FIG. 166.—*Case* 4. The same as *Fig.* 165, showing dilatation of third and lateral ventricles, and of foramina of Monro.

movement. The papillœdema was increasing. The urine was acid, contained no albumin, but there was a moderate amount of sugar and acetone.

The operation of decompression was about to be performed, but the patient's respiration failed during the shaving of the head; death ensued.

POST-MORTEM FINDINGS.—An endothelioma (meningioma) was found in the left frontal region and pressing on the Rolandic area. There was also marked hydrocephalus with pressure on the medulla.

FIG. 167.—*Case* 4. The same as *Fig.* 165, showing dilatation of the third ventricle and the aqueduct of Sylvius.

Case 5.—**Glioma of the cerebellum, secondary hydrocephalus, and signs suggestive of a supratentorial tumour.**

R. B., male, age 25 years, was admitted on Jan. 17, 1927, with a history of headache and vomiting for two months.

HISTORY.—Two months before admission the patient complained of bitemporal headaches and pain in the back of the neck. Speech became defective and was progressively worse before admission. Vision was progressively impaired and the gait became unsteady.

SUBSEQUENT PROGRESS.—Optic neuritis was evident one month after admission. Vomiting occurred occasionally every day, especially in the morning or at midday. A right subtemporal decompression was performed one month after admission, and a left subtemporal decompression one month later. Nothing abnormal was noted at the time of either operation.

The physical signs one week after the second operation were as follows. The right pupil was smaller than the left, and both reacted sluggishly. A divergent strabismus was present and the movements of all muscles supplied by the third nerve were weak. There was marked papillœdema of both optic discs, more obvious on the right side. There was a flaccid paralysis of the right side of the face and slight weakness of the left side. The patient refused to speak, but understood much of what was said. Hearing was diminished. The tongue protruded towards the right side. The condition of the sensory system was difficult to determine, but no gross loss was detected. A right-sided hemiplegia appeared after the second operation. In the upper limbs the tendon reflexes were more active on the right side than on the left. In the lower limbs the knee- and ankle-jerks were present and equal, ankle clonus was present on the right side, and the right plantar reflex resulted in dorsiflexion. The abdominal reflexes were absent on the right side. Incontinence of urine and fæces appeared after the second operation. The other systems were normal.

Death occurred fourteen weeks after admission.

POST-MORTEM FINDINGS.—The whole brain was extremely soft and, especially over the upper part of the cerebral hernia, breaking down and necrotic. After fixation the brain was cut in a series of horizontal sections and a large cerebellar tumour was found. The tumour was more or less centrally placed in the cerebellum, its lower part rather more to the right and its upper part slightly towards the left. It was soft, slightly pinkish-grey to ash-coloured, and fairly well-defined in its circumference, which was mostly in contact with and more or less embedded in distorted cortex. In the substance of the tumour there were a few small necrotic pseudo-cysts.

Microscopically, the tumour was highly cellular, the cells being 'undifferentiated' with small rounded to slightly oval nuclei, rich in chromatin and staining darkly, with a small to moderate amount of surrounding protoplasm, the outlines of which are very indefinite. The cells showed a distinct tendency to be arranged in irregularly rounded islets or alveoli, with thin-walled capillaries and scanty delicate connective tissue between them forming a scanty reticulum around these islets. According to Bailey and Cushing's classification, the tumour appeared to be a 'medulloblastoma'.

Case 6.—**Cerebellar arachnoid cyst, with secondary hydrocephalus and symptoms suggesting a lesion of the pyramidal tracts.** (*Fig.* 168.)

W. S., male, age 36 years, was admitted on Oct. 25, 1928, complaining of pain and stiffness in the back of neck.

HISTORY.—Three months prior to admission the patient began to have pain at the back of the eyes at the end of the day, after work. This pain had gradually

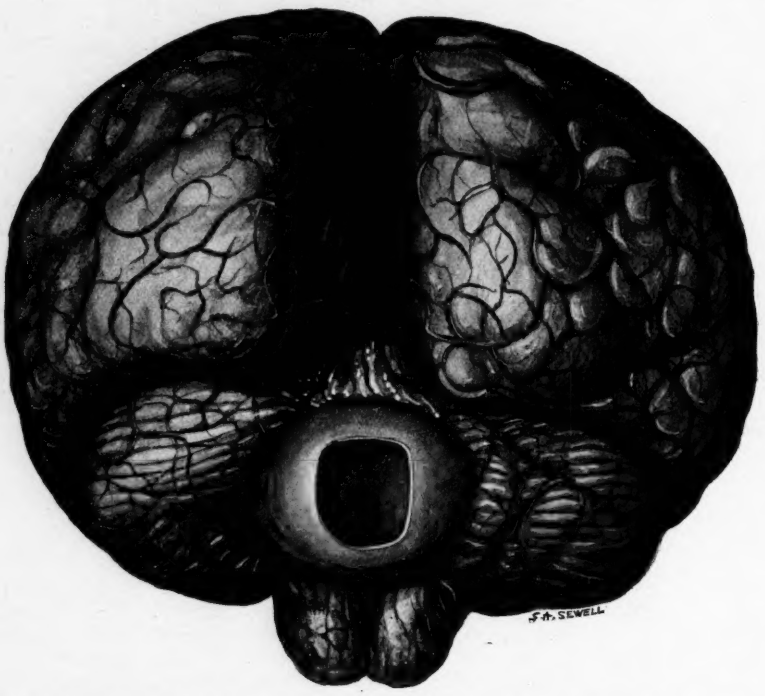


FIG. 168.—*Case* 6. Cerebellar arachnoid cyst with secondary hydrocephalus: showing cyst and flattening of the cerebral convolutions.

spread backwards, and at the time of admission it centred at the back of the neck and the muscles there seemed stiff. His previous health had been good.

ON EXAMINATION.—The pupils reacted to light and accommodation. Vision and the visual fields were normal. The optic discs on both sides were indistinct,

and there was a small hæmorrhage in the right retina. There were no nystagmus, sensory changes, or tremor of the hands, and the arm-jerks were normal. The knee-jerks were exaggerated, the ankle-jerks were present, there was a tendency to ankle clonus, and the plantar reflexes were extensor. The Wassermann reaction was positive in the blood and cerebrospinal fluid.

Subsequent Progress.—Oct. 27, 1928.—Lumbar puncture was performed and the fluid was under pressure. The patient stood it very well and there was no severe reaction.

Oct. 30.—The patient was allowed up in the afternoon as he said that the headaches, which were not relieved by lumbar puncture, were easier when he was up. He appeared as usual at night and slept well until 5.30 a.m. He then said he was not feeling well and looked tired. Five minutes later he was found propped up in bed, cyanosed, covered with cold perspiration, and quite unconscious. The breathing became feebler, the pulse was slow and weak, and the patient died within twenty minutes.

Post-mortem Findings.—The brain showed flattening of the convolutions and a convex bulge on the left side. Some of the surface vessels were obliterated. The cerebrospinal fluid was clear, and a large amount of fluid was present in the cerebellar fossa. The vermis of the cerebellum and the pons were compressed and flattened. In the middle line at the base of the cerebellum a space two inches in diameter was occupied by a thin-walled cyst. The arachnoid membrane in the region of the right auditory nerve was much thickened. The ventricles of the brain were distended with clear fluid. The cyst was diagnosed as a cerebellar arachnoid cyst.

Case 7.—**Glioma of the upper pons, midbrain, and interpeduncular space, secondary hydrocephalus and symptoms suggesting congenital hydrocephalus.** (*Figs.* 169, 170.)

J. P., male, age 6 years, was admitted on April 12, 1926.

History.—At the age of 5 months the patient was diagnosed as suffering from primary optic atrophy of both eyes. He complained of slight pain in the left leg about four months before admission. One month later he had a sudden attack of headache, vertigo, and vomiting. He was unable to walk after this attack, and trembling of the hands and knees appeared. His condition became steadily worse, his character changed, he became perverse and difficult to manage.

On Examination.—The pupils were equal and dilated, and did not react to light. The right eye was completely blind ; with the left eye he could count fingers. There was weakness of both external recti, and the eyes could not be elevated above the mid-horizontal line. Both optic discs showed secondary optic atrophy. There was difficulty in opening the mouth. There was weakness of voluntary movements of the face on the left side. The pulse was rapid. There was bilateral spastic paraplegia. The patient was unable to feed himself, and had incontinence of urine and fæces. He died on the seventh day after bilateral subtemporal decompression.

Post-mortem Findings.—The head was of distinctly large size as compared with the rest of the body. The skull was very thin, and both anterior and posterior fontanelles were still membranous. The convolutions of the brain showed definite flattening and pallor. A soft, tumour-like mass was found in the interpeduncular space, extending from the upper margin of the pons to well in front of the optic chiasma. The mass involved the chiasma, the tuber cinereum, and the infundibulum, and extended on each side to the tips of the temporosphenoidal lobes. The pituitary fossa was pressed upon, and was wider and shallower than normal. The pituitary body was pressed against the bottom of the fossa, and was saucer-shaped and widened. The posterior halves of the intracranial parts of the optic nerves were involved in the tumour ; the left being narrowed and degenerated, the right being soft, bulky, and œdematous. The sixth nerves appeared to be compressed against the bone by the tumour.

On horizontal section of the brain a marked degree of chronic hydrocephalus

was apparent, involving both the lateral and third ventricles. Embedded in the floor of the third ventricle there was a soft, rounded pinkish-brown tumour, projecting rather more to the right side than to the left. The tumour appeared to be cystic in nature, but, on cutting into it, it was found to be soft peripherally, and necrotic and hæmorrhagic in the centre.

Microscopically, the tumour was composed of small cells with dense, spherical nuclei, surrounded by a rather indefinite, feebly-staining cytoplasm. Between them

FIG. 169.—*Case* 7. Glioma of the upper part of the midbrain and the interpeduncular space in a child: view of brain and tumour from below.

was an indefinite material which did not stain for neurofibrillæ, neuroglia, or for connective tissue. Some of the small blood-vessels showed proliferation of their endothelial cells, sufficient in some cases to fill their lumen and form little solid masses. Both optic nerves showed thickening of their fibrous tissue and framework, with in the left advanced, and in the right complete, atrophy and disappearance of the nerve-fibres.

V. CLINICAL COMMENTARY.

The cases detailed above illustrate most of the points of interest in the diagnosis of cases in which secondary hydrocephalus is a complication and in the localization of the focal lesions present.

Case 1 was that of a man of 30 who for six weeks had suffered from head-

FIG. 170.—*Case* 7. The same as *Fig.* 169: the tumour and lateral ventricles viewed from above.

ache, vomiting, loss of vision, and vertigo. Physical examination revealed a series of signs—nystagmus, hypotonia, a tendency to fall towards the left—which seemed to point to a lesion of the left side of the cerebellum or adjacent to it. The symptoms continued and became more acute, the patient reeled as he walked, the head was carried forward on the chest, and the optic discs,

which at first were only blurred, in three weeks were found to have swollen to 4 dioptres. In addition there appeared analgesia in the ophthalmic and maxillary divisions of the left trigeminal nerve, and doubtful signs of a pyramidal lesion on the right side. A tumour of the posterior fossa of the skull was diagnosed and a subtentorial decompression advised. At the operation and at the subsequent autopsy it was found that the whole of the increase in intracranial tension was above the tentorium and that there was no increase in tension whatever below the tentorium. In reviewing the clinical features of the case the general symptoms were accepted as general symptoms of an intracranial neoplasm, and as such localizing signs as were present pointed to involvement of the cerebellum, a diagnosis was made of a tumour of or adjacent to the cerebellum, and treatment was advised accordingly. Subsequent review of the whole case showed that probably the more correct interpretation of the clinical features was as follows. The general symptoms were the result of increased intracranial tension above the tentorium as the result of hydrocephalus. The cerebellar signs were the result of secondary pressure upon the contents of the posterior fossa, and not to direct involvement of or pressure upon the cerebellum by the tumour itself. It is true that the diffuse gliosis of the pons was adjacent to the cerebellum, but it was not of such a character as to produce the cerebellar signs by direct pressure. The cerebellar signs were, in fact, false localizing signs secondary to the internal hydrocephalus and not true localizing signs. The case raises the question of the value of the localizing signs present, and of the correct interpretation of the so-called general symptoms of intracranial tumour.

Case 2 was that of a girl, age 4 years, who had been unable to walk for eighteen months and had had ataxia of the lower limbs only during that time. Because of the poor mentality of the parents certain important points in the history of the child were wanting. On examination the child was found to have an unusually large head, optic atrophy on both sides, varying signs of involvement of the pyramidal tracts, and ataxia of the lower limbs. Throughout the illness the mentality of the child was perfectly good, and excepting for a period of one week when she was unusually drowsy there were no symptoms directly suggesting the presence of an intracranial tumour. The conditions to be considered in diagnosis appeared to be a degenerative condition involving the pyramidal and cerebellar tracts, a chronic inflammatory condition involving these tracts and the optic radiations, and the effects of a previously undetected hydrocephalus. The congenital form of hydrocephalus was excluded because the development of the child had been apparently normal in every respect up to the age of 3 years, and the acquired form because there was no history of an inflammatory condition within the skull. With a view to clearing up the problem ventriculography was carried out. Internal hydrocephalus was proved to be present, but the cause of it was still unexplained. The alarming symptoms of the child immediately after the injection of air into the lateral ventricles and her sudden death some hours later raised the question of the advisability of performing ventriculography in such cases, of the best method to be employed, and of the cause of the accidents which sometimes attend this procedure. Subsequently autopsy showed that internal hydrocephalus was present, and was the result of

obstruction to the outflow of cerebrospinal fluid by a tumour of the inferior part of the cerebellum. In addition to the points in connection with ventriculography mentioned above, this case raised the question of differential diagnosis in the presence of hydrocephalus, and of the importance of a detailed history in the presence of this complication.

Case 3 was that of a patient, age 45, who had suffered from a nervous breakdown, loss of energy, mental irritability, an increasingly poor memory, headache, and vomiting for eight months. On examination the only physical signs present were inequality of the pupils, doubtful constriction of the visual fields, a slight increase in muscle tone on the right side of the body, and bilateral papillœdema more obvious on the left side. The general symptoms seemed to point to the presence of an intracranial tumour, and the abnormal mental state suggested that it was in the frontal lobe; while the difference in the muscle tone on the two sides with tremor and clumsiness of the right arm seemed to be in favour of a localization in the left frontal lobe rather than in the right. A decompression operation was consequently done over the left fronto-parietal region, and the patient died two days later of a subdural hæmorrhage. The autopsy revealed the presence of dilatation of the lateral ventricles more pronounced on the left side than on the right caused by a tumour of the upper part of the midbrain and the septum pellucidum. This case again raised the question of the significance of the general symptoms of intracranial tumour, and of the localizing value of the symptoms and signs usually thought to be associated with lesions of the frontal lobes. The indefinite localizing signs present in this case were again false localizing signs produced by secondary hydrocephalus.

Case 4 was an example of secondary hydrocephalus due to the vicious circle set up by a tumour so situated that it could not directly block the circulation of the cerebrospinal fluid. A patient who had been increasingly drowsy for three weeks and had shown mental symptoms for a few days was admitted in a state of coma. Following admission a number of interesting points were noted in connection with the clinical features: (1) The limbs of the right side were stiff, and there was some retraction of the head, but as the coma increased the limbs showed alternating flaccidity and rigidity, and finally became completely flaccid. (2) In the early stages the reflexes were all increased, but as the coma deepened they became less active and control of the organic reflexes was lost. (3) When the pulse-rate was 68 and the temperature subnormal the urine was perfectly normal; but, as signs of bulbar paralysis appeared, the temperature rose and glucose and acetone appeared in the urine. A further interesting point was the fact that a tumour, which must have been developing in a silent area of the brain for a long period, suddenly gave signs of increased intracranial tension, probably from indirect pressure on the medulla, and produced a fatal result within three weeks of the first indication of the presence of an intracranial condition. In this case prompt recognition of the presence of secondary hydrocephalus and adequate treatment of that condition could have led to the complete removal of the tumour itself. In addition to the points mentioned in connection with the previous cases, this case shows that secondary hydrocephalus due to a tumour not directly obstructing the circulation of cerebrospinal fluid

can arise *de novo*; it also raises the question of the significance of glycosuria as a symptom in cases passing into coma with only papillœdema to indicate that increased intracranial tension is present.

Case 5 was that of a patient who had bitemporal headache and pain in the back of the neck for two months. In addition the speech was progressively defective, and walking was becoming more and more unsteady. The signs of increased intracranial tension were present—namely, vomiting and papillœdema. The clinical features of the case at the time, not described in detail in this paper, appeared to point to a tumour of the frontal lobe as the cause of the symptoms; but, in view of the indefinite localization of the lesion and the urgency of the general symptoms, measures were adopted to relieve the increased intracranial tension. At the autopsy there was found a central tumour of the cerebellum obstructing the circulation of the cerebrospinal fluid and producing internal hydrocephalus. In this case the signs of a cerebellar lesion were apparently so indefinite, those of increased intracranial pressure so definite, and those of a lesion of the frontal lobe so suggestive, that a diagnosis was made of a frontal rather than of a cerebellar tumour. Closer attention to the sequence of events might have shown that the difficulty with speech and the impairment of gait were present from the onset of the clinical history, whereas the general symptoms were late in appearing and comparatively slow in developing—a sequence which would have suggested that more attention should be paid to the symptoms of a lesion of the posterior fossa, even though they were apparently indefinite. The physical signs which appeared after the two operations cannot be included in this discussion, as it is probable that they were, in some measure at least, dependent upon pressure effects in the neighbourhood of the operation areas. This case suggests, therefore, that in the presence of secondary hydrocephalus it is essential to make a careful study of the order in which the various symptoms and signs have developed, even in cases in which they are indefinite, in order to distinguish true and false localizing signs.

Case 6 was an example of a patient who had a quiescent arachnoid cyst of the cerebellum which finally produced secondary hydrocephalus. The chief points of interest in the case are the absence of symptoms of the focal lesion, and the slight indications of involvement of the pyramidal tracts apparently resulting from the secondary hydrocephalus alone.

Case 7 was that of a patient who had been affected since early infancy, and in consequence the clinical findings closely resembled those of the congenital form of hydrocephalus. At the age of 5 months he had been diagnosed as suffering from primary optic atrophy. From that age until three months before death the clinical picture was that of a slight degree of congenital hydrocephalus. At that time he had a sudden attack of headache, vomiting, and vertigo, after which he was unable to walk, and trembling of the hands and knees appeared. At that stage there appeared certain physical signs which indicated that there was a lesion involving the midbrain, and it was also recognized that the changes in the optic discs were characteristic of secondary and not of primary optic atrophy. At the autopsy there were found changes characteristic of secondary hydrocephalus involving the third and the lateral ventricles; also a tumour mass in the floor of the third

ventricle and obstructing the circulation of the cerebrospinal fluid below that level. The remarkable feature of the case was that, apart from the optic atrophy, there were no general symptoms of increased intracranial pressure for over five years, while definite localizing signs of the tumour appeared only after the increase in intracranial pressure had been present for that time. The case illustrates the similarity of the clinical features of secondary hydrocephalus arising as the result of a tumour in infancy to those of congenital hydrocephalus; also the adaptation of the skull of the child to the increasing pressure to such a degree that, apart from the optic atrophy and the impairment of vision, no general symptoms of increased intracranial pressure were present except during the short attack three months before death.

VI. SECONDARY HYDROCEPHALUS AS A FACTOR IN DIAGNOSIS.

The symptomatology of secondary hydrocephalus *per se* would appear to be much more extensive than is usually thought. In general that of acquired hydrocephalus differs considerably from that of the congenital form of the disease. When, however, the condition arises very early in infancy or even in early childhood, as in *Cases* 2 and 7, it may closely imitate the congenital form. Enlargement of the head, a cracked-pot sound on percussion, and separation of the sutures may be present in secondary hydrocephalus which has begun in early childhood.

The statement frequently quoted in text-books that the symptoms of hydrocephalus caused by brain tumour are essentially those of the underlying condition is apt to be misunderstood. It would probably be more correct to say that cases of cerebral tumour, both those in which focal signs have been present and those in which they have been absent, are liable to develop symptoms of secondary hydrocephalus. In the former group the appearance of focal symptoms followed by general symptoms at once leads to the correct diagnosis of an intracranial tumour; but in the latter group such a diagnosis would be purely presumptive. Conditions other than an intracranial tumour may lead to the development of an intracranial state which produces all the general symptoms present in cases of tumour. These general symptoms—headache, vomiting, vertigo, and increasing papillœdema—are essentially the symptoms of increased intracranial tension, and those which may occur with secondary hydrocephalus from any cause. Each of them alone may be present as a result of increased intracranial tension, or as a symptom of other conditions to be referred to later.

When localizing symptoms *appear after the development of general symptoms*, they may be either *true* or *false* localizing signs, and a careful review of the clinical features of the case is necessary in order to estimate their true value in diagnosis. Such false localizing signs may be: general constriction of the visual fields, paralysis of lateral deviation of the eyeballs on one or both sides, mental symptoms suggesting a lesion of the frontal lobe, unilateral or bilateral symptoms suggesting slight interference with the pyramidal and sometimes the cerebellar tracts, symptoms and signs suggesting a lesion of the cerebellum, signs of pituitary dysfunction, unilateral or bilateral deafness, and tinnitus.

It is evident, therefore, that in the presence of the general symptoms of intracranial tumour arising *de novo* it is unsafe to diagnose an intracranial tumour on them alone without further investigation to determine the cause of the increase in intracranial tension; and, further, that it is unsafe to rely upon localizing symptoms which have developed subsequently in making such a diagnosis and in localizing the lesion unless they are beyond all doubt and are supported by other signs which place the lesion in the same position.

The differential diagnosis in a case showing the general symptoms which may be present with secondary hydrocephalus is a matter of some importance. Souttar[7] has stressed the importance of the differential diagnosis in cases associated with increased intracranial tension from such general conditions as renal disease, severe anæmia, and lead poisoning. In cases in which the tumour is placed in the frontal lobe and there is pressure either direct or indirect on the medulla, the differential diagnosis from diabetes mellitus may cause difficulty. In *Case* 4 the only method of differentiation between diabetes mellitus and increased intracranial tension during the later stages was the presence of papillœdema, and the same difficulty arose in connection with one of the cases reported by Bingel.[8] The cerebral form of disseminated sclerosis, in which retrobulbar neuritis is placed so far forward as to involve the optic disc, may cause difficulty in some cases; while the rare cases of papillœdema with transverse myelitis may cause confusion when the condition of the optic disc appears before that of the cord. Such general conditions are, however, readily excluded by a careful general examination of the patient.

Once it has been established that the general symptoms are the result of increased intracranial tension, it is necessary to decide whether they are due to massive tumour growth, œdema of the brain, general circulatory changes, or internal hydrocephalus. In those cases in which one or more localizing signs have been followed by the general symptoms of increased intracranial tension, a satisfactory working diagnosis is usually made at this stage, and, according to Russell Brain,[6] the presence of hydrocephalus may be suggested by early and frequent vomiting and the rapid development of papillœdema. When, however, there have been no localizing symptoms before the onset of general symptoms and the presence of secondary hydrocephalus is suspected, the problem is to prove that it is present, to determine the site of the lesion, to give a reasonable opinion as to its nature, and to advise the correct treatment. Ventriculography is, as a rule, the most useful method available for establishing the diagnosis and localizing the lesion; but it is not always advisable to use it. Penfield[9] recommends that this method should be avoided when there is much increased intracranial tension, but goes on to say that if the diagnosis of increased intracranial tension is certain, "air injection is of the greatest assistance and should prevent many negative explorations: it should therefore be used without hesitation in such cases" with the object of demonstrating the presence or absence of a tumour, and of determining the size and position of the ventricles. Grant[10] states that in 392 cases examined by ventriculography, that method of examination was of value in localization in 311 (79·3 per cent); while in 93 cases (23·0 per cent of the total) the lesion was localized on ventriculography alone. Of the latter, 44 cases (11·2 per cent of the total, or 40·0 per cent of the 93 cases) were

amenable to surgical treatment—figures which dispose of the idea that it is only the deep-seated inoperable tumours which are identified by ventriculography alone.

Having decided that a blockage to the outflow of cerebrospinal fluid is present, it is necessary in view of the treatment to be adopted to decide what is the cause of the blockage. Dandy and Blackfan[11, 12] showed that hydrocephalus might be: (1) Obstructive, due to congenital malformation, an inflammatory process, or to a tumour in any part of the ventricular system, but usually at the aqueduct of Sylvius, the foramen of Magendie, or the foramina of Luschka; or (2) Communicating, due to a barrier of adhesions at the base of the brain preventing the cerebrospinal fluid circulating through the cerebral subarachnoid space. Such adhesions are usually the result of meningitis occurring either before or after birth, and frequently so mild as to be overlooked. This was pointed out by Hilton and of later years by Fraser and Dandy,[13, 14] who stressed the fact that not only post-inflammatory adhesions in the cisterna magna, but also congenital maldevelopment of the subarachnoid space, tumour of the brain-stem, and abscess in the same region acted similarly. Young[15] reported a case in which the clinical history was very suggestive of brain tumour, but in which hydrocephalus was present and disappeared after eight years. In some of the cases of hydrocephalus developing as a result of the above conditions a detailed history may be of considerable value in determining the presence of post-meningitic adhesions or abscess, while a history of focal symptoms before the development of general symptoms may serve to establish a tumour as the cause of the blockage. But when all the clinical methods have been employed there will still remain a proportion of cases in which there is no clue to the nature of the condition, and it is in these that the use of ventriculography is particularly necessary if the patient is to have his chance.

The risk of withholding this method of examination is much greater than that of using it, and, in view of Grant's[10] figures, it seems perfectly justifiable to use it even in face of the apparent risks in order to diagnose and localize the proportion of lesions which are amenable to surgical treatment in such cases.

VII. SECONDARY HYDROCEPHALUS AS A FACTOR IN LOCALIZATION.

It has been pointed out above that all localizing symptoms which develop after the appearance of general symptoms of increased intracranial pressure must be regarded with suspicion; and that they may be used for localizing the lesion only if they are clear and unmistakable evidences of a focal lesion.

Of these false localizing signs, those suggesting involvement of the cerebellum are probably the most common. Nystagmus, hypotonia, intention tremor, and other symptoms of a cerebellar lesion may be present. They were present in *Case* 1, but were never sufficiently definite to make a diagnosis of a cerebellar lesion on them alone. They have been referred to by other writers. In Young's[15] patient fine nystagmus on lateral deviation, hypotonia of the limbs, and intention tremor were present, and disappeared on the

recovery of the patient. Two more definite cases were reported by Bramwell, [16, 17] both of internal hydrocephalus in which cerebellar signs were pronounced—in one of them the result of posterior basilar meningitis and in the other the result of obstruction at the foramen of Magendie by adhesions. Spiller[18] recorded a case of hydrocephalus with cerebellar signs in which at autopsy only hydrocephalus due to obstruction and closure of the aqueduct of Sylvius was found. Oppenheim[19] recognized the close association of the clinical signs of brain tumour and internal hydrocephalus in two cases, in one of which cerebellar signs were present, and at autopsy only internal hydrocephalus was found. Rhein[20] reported a case with cerebellar signs in which there were cystic dilatations of the lateral recesses of the fourth ventricle in the neighbourhood of the cerebellopontine angle. These examples of cerebellar signs appearing in cases of hydrocephalus are sufficient to show that, under such conditions, cerebellar signs which are not absolutely definite and develop with or after the appearance of general symptoms of increased intracranial tension are false localizing signs, and, as such, are of no value whatever in determining the site of the lesion.

In *Case* 3 the symptoms were strongly suggestive of a lesion of the frontal lobe of the brain. For eight months the patient had had a 'nervous breakdown', and had suffered from headache, vomiting, irritability, loss of memory, and loss of energy. On examination it was found that her mental condition was facile, there was a certain degree of euphoria, the recent memory was defective, and the facetiousness and jocularity or 'Witzelsucht' mentioned by Oppenheim and other writers was very definite. There was no focal lesion whatever present in the frontal lobes, and it was evident that the internal hydrocephalus was entirely responsible for the symptoms mentioned. Russell Brain[6] mentions mental deterioration and irritability as common symptoms of cerebral tumour complicated by hydrocephalus, and stated that in one case dysphasia was present. Though never very definite as localizing signs of a frontal lobe lesion, it is obvious that such symptoms must always, in the presence of unmistakable evidence of increased intracranial tension, be looked upon with suspicion and regarded as false localizing signs.

In this case the erroneous localization in the frontal lobe was supported in some measure by the fact that there were slight indications of involvement of one pyramidal tract, probably, as it proved, because one foramen of Monro was obstructed before the other and the corresponding lateral ventricle more distended than that on the other side. Though in this case the signs were quite indefinite, it is apparent that minor signs of a pyramidal lesion, upon which in obscure cases much reliance is apt to be placed in the attempt to determine the side of the lesion, may also be false localizing signs as a result of secondary hydrocephalus. Similar signs were also present in *Case* 6. The indications of a lesion of one pyramidal tract were more definite in one of the cases reported by Oppenheim.[19] In this case there were bilateral optic atrophy, contracture of the sternomastoid and trapezius, weakness of the left leg, an extensor plantar reflex on the right side, and exaggerated deep reflexes. Russell Brain[6] also includes involvement of the pyramidal tracts as a result of hydrocephalus and characterized by weakness, spasticity, and bilateral extensor responses as a feature of hydrocephalus complicating brain tumour.

Signs of pituitary dysfunction may appear in the course of long-standing hydrocephalus and lead to the suspicion that the pituitary gland is primarily at fault. In *Case* 1 concentric contraction of the visual fields was present even before the signs of papillœdema were evident. In *Case* 2 the child had become unusually obese since the onset of the illness, during which internal hydrocephalus had evidently been present for from eighteen months to two years. In *Case* 3 there was again, as far as could be determined, definite concentric contraction of the visual fields—a finding which led to the suspicion at one stage that a suprasellar tumour might be the cause of the symptoms. In this case, however, the position of the tumour suggested that the contraction of the visual fields might not be due entirely to hydrocephalus, but *Case* 1 suggested that the contraction might be due to the hydrocephalus alone. More definite accounts of the occurrence of the signs of pituitary lesions in the course of hydrocephalus have been recorded in the literature. Jacobaeus[21] reported the case of a female, age 20 years, who suffered from dystrophia adiposogenitalis, and had choked discs and other signs of increased intracranial tension. By means of ventriculography the diagnosis of hydrocephalus was established, and at the autopsy it was found that the only pathological condition present was internal hydrocephalus due to absolute obstruction of a chronic inflammatory nature in the aqueduct of Sylvius. In Young's[15] patient, at the age of 16, after hydrocephalus had been present for eight years, the weight was 162 lb., the sugar tolerance was slightly increased with a raised threshold, and the skiagram showed the presence of hydrocephalus and an enlarged sella turcica. Cushing[22] reported several cases in which hydrocephalus was associated with posterior-lobe insufficiency, and one in which there were acromegalic features. Marienesco and Goldstein[23] reported two cases of hydrocephalus with adiposity of the female type and genital hypoplasia, one with a cyst of the cerebellum and the other of unknown etiology. Kurt Goldstein[24] reported three cases of hydrocephalus in which changes in the sella turcica were noted together with obesity and under-developed genitalia; and similar cases have been reported by Stumpf,[25] Pollock,[26] Strauch,[27] and Schultz.[28] In Russell Brain's[6] 60 cases of cerebral tumour there were two which showed adiposity and infantilism of the Frölich type. It is evident, therefore, that in recent cases of hydrocephalus with headache, vomiting, vertigo, and papillœdema, the presence of bilateral constriction of the visual fields may lead to the erroneous conclusion that a suprasellar tumour is responsible for the condition; and that, in cases of longer standing, the signs usually associated with pituitary dysfunction, including changes in the sella turcica itself, may appear probably as a result of constant pressure on the hypothalamic region and the infundibulum.

Bilateral deafness was present in *Case* 2 and has been associated with tinnitus in several cases observed by us. In *Case* 2, in particular, it was found necessary to exclude the possibility of an intracranial complication of middle-ear disease before considering other explanations of the patient's condition.

Thus cerebellar signs, evidence of lesions in the frontal lobe, changes in the visual fields, minor signs of involvement of the pyramidal tracts, and signs of pituitary dysfunction and chiefly hypofunction, minor or fully-developed according to the duration of the condition, may appear in the

course of secondary hydrocephalus due to obstruction to the outflow of cerebrospinal fluid from any cause. Certain other signs occurring in the course of hydrocephalus and liable to be interpreted as localizing signs will be mentioned in the account of the clinical investigation of the condition. These signs are false localizing signs and, in cases in which the diagnosis of internal hydrocephalus is established or even suspected on the symptoms present, must be carefully considered and their time relations to the general symptoms determined before they can be used in the localization of the primary lesion.

VIII. THE INVESTIGATION OF SECONDARY HYDROCEPHALUS AND THE RESULTS OBTAINED.

The investigation of a case in which secondary hydrocephalus may be present is carried out by: (1) *Clinical methods—that is, a careful and detailed examination of the central nervous system;* (2) *Puncture methods, including lumbar puncture, cisternal puncture, and ventricular puncture;* (3) *X-ray examination;* (4) *Ventriculography; and* (5) *Observations made at the time of the operation.* These methods of examination are considered in turn, the clinical features which may be present in association with secondary hydrocephalus are described both as regards their value as symptoms of the condition and in relation to the possibility of confusion with those of focal lesions, and the indications both for and against the more technical methods of examination are detailed.

1. CLINICAL METHODS.

The symptoms of secondary hydrocephalus may appear secondary to those of a local lesion of the brain or of the cranial cavity, or may arise *de novo* in a patient who has been previously in good health. The chief symptoms of secondary hydrocephalus are essentially those which are usually regarded as the general symptoms of a cerebral tumour. They are primarily the symptoms of increased intracranial tension and may be due to conditions other than hydrocephalus. There are, however, certain symptoms and signs which appear earlier or more frequently in the presence of hydrocephalus, and serve in some measure to differentiate that condition from the other causes of increased intracranial tension.

The general symptoms of secondary hydrocephalus are well known. Headache is probably the most frequent symptom, but its severity depends upon the rapidity with which the obstruction to the circulation of the cerebrospinal fluid develops, and upon the non-yielding character or otherwise of the skull itself. After it has once appeared it seldom disappears completely, though it is subject to considerable fluctuations. It is usually described by the patient as a dull ache, or more often as a dull sense of pressure within the head, associated with a slight sense of physical ill-being or nausea which prevents him fixing his attention upon any occupation or recreation for any length of time. It is varied at times by acute exacerbations, during which the patient prefers to lie still and resents being disturbed. During these exacerbations it is aggravated by visual or auditory stimulation, or by anything which tends to increase the intracranial tension even for a

short time, such as stooping, running, coughing, or straining. It is frequently increased when the vomiting is most severe, probably for the same reason. The severity of the headache and the frequency and intensity of the exacerbations depend upon the rapidity with which the hydrocephalus develops. When developing suddenly in an adult the pain may be agonizing until some obscuring of the mental faculties appears. When developing in a young child, as in *Cases* 2 and 7, there may be nothing more than a feeling of malaise, sometimes associated with vomiting and vertigo, for a week or more, and then the child is again apparently well. The particular incidence of the acute exacerbations during the night and on awakening in the morning has not, in our experience, been definite enough to be of any value in diagnosis. Russell Brain,[6] however, states that in his cases the headache was especially severe at these times, and explains that this particular incidence is due to the fact that the blood-pressure, and consequently the intracranial pressure, falls during sleep and rises again in the latter part of the night and on awakening.

Vertigo is often associated with the headache, and, in our experience, has been more frequent in the presence of hydrocephalus than of œdema of the brain or circulatory changes complicating intracranial tumour. It is not a true vertigo of the type usually associated with cerebellar or labyrinthine dysfunction. It is rather a light-headedness or a swimming in the head, and is often described by the patient as such. It gives him the feeling that he is uncertain of his position, that he must be careful how he moves his limbs or where he puts his feet if he is to avoid falling; and causes him to look for support when he attempts to move from place to place. Of staggering from side to side, or a tendency to fall in one direction or the other, there is usually little evidence until minor cerebellar symptoms develop as a result of direct pressure from above the tentorium. These symptoms will be discussed at a later stage.

Vomiting is a frequent symptom which also varies with the rapidity of the onset and is subject to exacerbations and remissions. In the adult it is usually most severe at the times when the headache is severe. Russell Brain[6] points out that it may be due to hydrocephalus alone, and, rejecting the explanation that it may be due to pressure on the medulla or to irritation of the vagus, believes it to be a reflex act of which the appropriate stimulus is a rise in the intraventricular tension. The vomiting is apt to be more frequent and more severe when the patient assumes the erect position, but at such times can often be lessened if the change in position is made gradually. It may or may not be preceded by extreme nausea, but it is often more frequent and more severe in those patients who complain of a constant feeling of nausea. It is usually an early and severe symptom of secondary hydrocephalus, and is often of great importance in drawing attention to the onset of the condition. In a young child, however, in whom the skull may adapt itself to the increase in pressure, it may be present for only a short time and then disappear (*see Cases* 2 and 7).

Transient disturbances of vision are generally present. These are usually the appearance of spots in front of the eyes, sudden dimness or transient loss of vision, a blurred outline to objects, and a feeling that a dark wall is shutting the patient in on each side. They are often associated with exacerbations of

the headache and vertigo, and appear on changing the position or on sudden movement. The acuity of the vision is, as a rule, unimpaired at first; but the patient is often unwilling to do anything requiring the use of the eyes, as he feels that it will aggravate his general symptoms.

Mental symptoms of all degrees may appear quite apart from involvement of any particular part of the brain, and are apt to be confused with those which arise as a result of a focal lesion in the frontal lobe. Loss of energy and a constant feeling of fatigue are frequent symptoms in those patients in whom the hydrocephalus has developed slowly (*Case* 3). There may be mental irritability for a time, but, as a rule, inability to concentrate and to maintain attention are the important features. The memory is often defective, that for recent events is particularly affected, but later the remote memory and the time relations of past events are usually confused. There is increasing inability to carry on an occupation or to attend to any duties, and consequently such patients are often misunderstood or thought to be suffering from a 'nervous breakdown' before the true condition is discovered. Such misunderstanding is apt to arise when the symptoms develop slowly (*Case* 3), but does not occur when more characteristic symptoms of increased intracranial tension develop rapidly (*Case* 1). The confusion of hydrocephalus with a lesion of the frontal lobe is often increased by the presence of an unnatural friendliness or a characteristic euphoria on the part of the patient. He may talk easily but wander from one subject to another, may show complete mental detachment, utterly disregard his physical condition, and, far from being disturbed by it, may feel and show others that he is capable of performing even the most difficult tasks. In fact, he may show all the symptoms usually associated with early dementia paralytica. A prominent symptom in one of our patients was a tendency to jocularity in every word that was spoken; and in two of them an unnatural willingness to have anything done to them as long as it was done quickly. Papillœdema, euphoria, and a steppage gait, all of which disappeared after decompression of the posterior fossa, were associated with dilatation of the ventricles without a tumour of the frontal lobe in a man of 40 referred to by Laignel-Lavastine and Cl. Vincent.[29] In some patients stupor is more obvious, especially when, for the time being, the intracranial tension is above the usual level. In one patient stupor always disappeared on the removal of a small quantity of cerebrospinal fluid, but recurred after sixty hours. As the pressure increases, the patient becomes more stuporose and may sink into a coma.

Generalized convulsions are distinctly unusual in the course of secondary hydrocephalus. They are mentioned by Russell Brain[6] as a symptom of hydrocephalus complicating cerebral tumour. It would appear that, with the increase in intracranial tension, the tendency to the release of function or the irritation of motor cells is lessened rather than increased, and it has been observed in cases of hydrocephalus supervening on a focal lesion characterized by convulsive seizures that the tendency to attacks diminished as the pressure increased. On the other hand, in certain cases, notably that of Gordon,[30] seizures have been present with an increase in pressure, and have become less frequent or disappeared on the relief of pressure.

The temperature is usually regarded as subnormal when the intracranial

tension is increased, but there are many exceptions to this rule. In three patients in whom a sudden increase in intraventricular tension had occurred, one of them following ventriculography, a sudden rise of temperature to 104° was observed, and in none of them could any other cause of the rise be found. It is possible that the rise in temperature in these cases is comparable with that which often follows intraventricular hæmorrhage. In one of our patients an inexplicable symptom was a subjective feeling of chilliness which had been present from the onset of the condition.

The pulse-rate is usually normal, and it is only in the terminal stages when the patient is sinking into coma that a slowing of the rate occurs. The respiration-rate does not appear to be appreciably affected during the active stage of the condition before indications of direct pressure upon the contents of the posterior fossa have appeared.

In short, there appears to be some justification for arriving at the following conclusions about the symptoms as opposed to the physical signs of uncomplicated secondary hydrocephalus: (1) When of sudden onset, the characteristic symptoms of increased intracranial tension appear rapidly in a severe form. (2) When of slow onset, more general symptoms such as those of mental changes may be present and symptoms of increased intracranial tension be less severe. (3) When developing in a young child, minor symptoms may appear for a short time and then disappear as the skull adapts itself to the increased pressure.

In addition to the symptoms, the physical signs of intracranial conditions are varied considerably by the presence of secondary hydrocephalus, and these variations appear not only in the form of alterations in motor power and reflexes, but also in the form of false localizing signs. The former group is best considered in the order in which the variations are encountered in the course of routine physical examination.

In the adult the skull presents no abnormality in shape or size, but occasionally some hyperalgesia of the scalp or tenderness on light percussion of the skull is present. This hyperalgesia is, as a rule, present generally over the vault of the skull, but occasionally it is confined to one side or the other or to the nape of the neck. In the young child the condition of the skull is that seen in the congenital form of hydrocephalus, and there may be great difficulty, apart from the history, in deciding whether a congenital or an acquired condition is responsible for the hydrocephalus (*Case* 7). The skull is enlarged and its circumference may approximate closely to that of the chest even in a child of 4 years, but, owing to the fact that facial structures have had a full opportunity of developing, there is not the same overhanging appearance of the forehead seen in the congenital form. Whether the sutures and fontanelles are affected depends upon the age at which the condition began. In *Case* 2 they were closed, in *Case* 7 they were represented by membranous tissue. The hyperalgesia and tenderness sometimes present in adult cases are usually absent. A cracked-pot sound may be present on percussion if the increase in pressure has been of long enough duration to thin the bones of the cranial vault.

The examination of the eyes presents many features of interest. Defective lateral deviation of the eyeballs may be present on one or both sides

entirely as a result of the general increase in pressure; and, associated with this sign, there may be transient diplopia and slight, irregular jerking movements of the eyeballs on looking to one side or the other at command, but not sufficiently definite to be classed as true nystagmus. The vision is good in the early stages, but when diminished, as it often is as the condition progresses, should lead to the suspicion that optic atrophy is already developing. In the early stages the retinal veins are often distended. The margins of the optic discs may be blurred and papillœdema develops rapidly. In the course of two or three weeks it may reach 5 dioptres and be associated with small hæmorrhages into the retina around the disc margins. If the condition persists or the increased intracranial tension is unrelieved, secondary optic atrophy rapidly develops, with failure of vision, pallor of the optic discs, obliteration of the physical cup, blurred disc margins, increase in pigment around the margins of the discs, and the presence of visible lymphatic sheaths along the vessels close to the disc. The appearance of papillœdema on one side and not on the other, or a greater degree of swelling on one side, does not necessarily indicate that the lateral ventricle on that side is distended more than that on the other side. In connection with this point, however, a comparison of *Cases* 1 and 3 is instructive.

Deafness and tinnitus may be present. There may be noises in the head which the patient describes as like the sound of a waterfall, preventing him hearing properly. Beyond a slight tendency to a lack of expression of the face in repose, the other cranial nerves are usually unaffected. Quite apart from the lack of co-operation due to the impairment of the patient's mental state, all types of sensation may be appreciated somewhat less readily than by the normal individual.

In the absence of evidence of involvement of the motor paths, the muscle tone is often reduced, motor power is slightly decreased, and the tendon reflexes are less active than normal. Cases have been observed in which it was impossible to elicit the tendon reflexes on one side, while on the other they could be elicited only because there was slight involvement of the corresponding pyramidal tract. The plantar reflexes are flexor, or great difficulty is experienced in eliciting any response; occasionally they are found to be extensor. There may be a slight, irregular tremor on one or both sides, but the cause of this sign is not at all evident. The tremor may, however, be confused with that which sometimes occurs with lesions of the frontal lobe, and has been suggested as a useful sign in localization in such cases. Beyond a slight unsteadiness in movement, both with the eyes open and with them closed, co-ordination is usually fairly good.

When the condition is severe and producing mental impairment, or when it has been present for a long time, there may be intermittent or permanent incontinence of fæces and urine. It was particularly noticeable in one case how lack of control of the sphincters appeared as the pressure increased, while complete control resulted as soon as the pressure was relieved.

The more definite false localizing signs which sometimes appear in cases of secondary hydrocephalus have already been referred to in discussing the condition as a factor in localization. The signs which suggest slight involvement of the cerebellum are particularly important, and may occur on one side

more than on the other or equally on the two sides. As a rule, however, these signs are rarely as definite as those which occur with a focal lesion of the cerebellum; nevertheless they are sufficiently suggestive in a case in which localization depends upon minor indications to be a source of confusion. More definite vertigo is present, the gait is often reeling, and the patient may feel a tendency to fall backwards or to one side. Nystagmus may be more definite, the speech may be slightly slurred, and the muscles of the limbs hypotonic. The motor power in the limbs is decreased, the tendon reflexes are more difficult to elicit, but vary from time to time, there may be some tremor of the hands on movement towards an object, a slight rebound phenomenon may be present, there may be a suggestion of decomposition of movements, and slight spontaneous deviation of the hands may occasionally be apparent. It will be recognized that, when occurring bilaterally, these signs are simply suggestive of cerebellar dysfunction and no more; but when occurring on one side more than on the other, it will be obvious that they will lead to difficulties in a case in which one is forced to depend upon minor variations in attempting to localize the lesion. In *Case* 1 they were present more on the left side than on the right. Nystagmus was definite with a long, slow swing to the left, there was a tendency to fall back and to the left, there was a tremor of the left hand on movement, the slight rebound defect was more obvious on the left side than on the right, there was slight spontaneous deviation of the left hand to the left, and the tendon reflexes were more difficult to elicit on the left side than on the right—all signs which suggested, but were not definitely diagnostic of, a lesion of the left side of the cerebellum. As, in this case, the main pontine lesion was very small and did not exert any direct pressure upon the cerebellum, it was evident that the symptoms depended largely upon the secondary hydrocephalus present. There was one feature, however, which may serve to distinguish between primary and secondary cerebellar signs—namely, that the signs varied from day to day and would be definite one day and absent the next, an extreme variability of signs which is not usually observed in true cerebellar lesions.

The mental symptoms which may suggest a lesion of the frontal lobe have already been referred to earlier in this discussion. Minor indications of a pyramidal lesion may appear on one or both sides, and it would seem that they are more likely to arise when the development of the hydrocephalus has been rapid. Some increase in the muscle tone of the limbs may appear together with an increase in the deep reflexes as compared with the normal for the patient. When such slight variations occur more on one side than on the other, or on one side only, it appears that the signs correspond to a greater distension of the lateral ventricle on the opposite side. These signs are comparable with the general muscular rigidity, sometimes more definite on one side than on the other, which may occur with intraventricular hæmorrhage, and are probably less obvious because of the slower rate of development (*Case* 4). Cases have been seen in which more definite indications, such as inequality of the abdominal reflexes and a transient extensor plantar reflex, were present. The evidences of dyspituitarism developing in the course of secondary hydrocephalus are particularly interesting, but it is evident that they are liable to occur only in those cases of gradual onset and of comparatively long duration.

2. PUNCTURE METHODS.

The puncture methods which may be employed in the investigation of secondary hydrocephalus are lumbar puncture, ventricular puncture, and ventricular puncture with estimation of the position and volume of the lateral ventricles. Cisternal puncture is rarely, if ever, used, except incidentally, in the investigation of this condition.

As a rule the cerebrospinal fluid withdrawn by lumbar puncture shows no abnormality, but if there is a complete obstruction of the communicating channels between the lateral ventricles and the spinal subarachnoid space, very little fluid may be obtained by this means. In *Case* 1 the cerebrospinal fluid was normal except for the following Lange curve—0021000000. In *Case* 3 the cerebrospinal fluid was again negative and produced the same Lange curve. In *Case* 2 at the first examination there were 4 'small lymphocytes' per c.mm., the Lange test was negative, and the protein was unusually low (0·0075 per cent). At a subsequent examination, there were 8 'small lymphocytes' per c.mm., the protein was again low (0·006 per cent), and there was a slight excess of globulin. On this occasion the Lange curve was the same as that in *Case* 1. In *Case* 6 the cerebrospinal fluid was normal; while in *Case* 4 it was not under pressure, was clear, contained 10 lymphocytes per c.mm., and 0·1 per cent of protein.

Beyond noting that in *Case* 3 the fluid withdrawn from the ventricles showed no increase in cells and was unusually poor in protein, we have had no opportunity of investigating the condition of the fluid withdrawn from the ventricles.

It is possible that direct ventricular puncture with the measurement of the pressure of the fluid in one or both lateral ventricles, and a comparison of the intraventricular pressure with that obtained on lumbar puncture or cisternal puncture, may serve to establish the presence of a block in the circulation of the cerebrospinal fluid between the two ventricles, between the ventricles and the spinal subarachnoid space, or between the ventricles and the cisterna magna. Dandy[31] has employed an ingenious method of ventricular estimation for the localization of brain tumours in comatose patients. He taps both lateral ventricles and estimates their size and position. If both are dilated and they are in communication as shown by indigo-carmine, he concludes that the tumour is in the posterior fossa and explores in that region. If one is dilated and the other collapsed, he explores on the side of the collapsed ventricle.

It is evident, therefore, that, apart from the information to be obtained by ventricular estimation, the examination of a patient suffering from secondary hydrocephalus by puncture methods usually produces evidence of a negative character.

3. X-RAY EXAMINATION.

As a rule in the adult case a skiagram gives little information of value apart from the evidence of increased vascularity in the neighbourhood of a meningeal tumour or endothelioma. In the cases which have begun in early childhood, however, the enlargement of the cranial vault may be confirmed, thinning of the bones of the vault may be noted, the bones may present a

beaten silver appearance, and accentuation of the vascular grooves may be apparent. The last-named feature may also be noted in adult skulls if the intracranial pressure has remained high for a long period.

Certain changes in the base of the skull may be seen in cases of long standing, and especially in those associated with pituitary dysfunction. Decrease in the overhanging clinoid processes, flattening of the sella turcica from above down, and even enlargement of the cavity itself may be present. The possibility that such changes as these may be present in cases of secondary hydrocephalus in association with symptoms of pituitary dysfunction, constriction of the visual fields, and papillœdema is of importance in diagnosis and localization, for a group of findings of this nature is likely to suggest that a lesion of the pituitary gland itself or of the suprasellar region is entirely responsible for the clinical picture, whereas the occurrence of pituitary dysfunction and changes in the sella turcica secondary to hydrocephalus may be overlooked. It would appear that these changes are brought about by direct pressure either from within or without the gland, for the wide dilatation of the infundibulum itself would suggest that pressure may be exerted from within the gland as well as from without.

Irrespective of the relation of intracranial tumours to secondary hydrocephalus, Cairns[32] has pointed out the value of X-ray examination in the diagnosis and localization of such tumours. The presence of calcification in 71 per cent of craniopharyngeal pouch cysts according to McKenzie and Sosman,[33] the positive radiological findings in 'meningiomata' described by Sosman and Putnam,[34] evidence of calcification in 10 per cent of gliomata according to Van Dessel,[35] and evidence of displacement of the falx cerebri or of the pineal body to one side of the middle line by noting the position of shadows of calcified areas in one or the other, show how X-ray examination may be of considerable value in the diagnosis and localization of the lesions giving rise to secondary hydrocephalus.

4. VENTRICULOGRAPHY.

Having reached the stage in the investigation of a case in which a tumour is suspected to be present and possibly complicated by secondary hydrocephalus, it is necessary to decide whether ventriculography should be employed to confirm the diagnosis and, if possible, to localize the responsible lesion. This resolves itself into the consideration of the indications for and against employing the method, the technique of the method to be used, the sites of puncture, the positions in which the head is to be photographed, and the interpretation of the films themselves.

It cannot be denied that this method of examination exposes the patient to certain risks, and these must be carefully considered and weighed against the condition of the patient and the value of the information that may be gained. In *Case* 2, towards the end of the operation, the breathing became stertorous, vomiting occurred, and the patient went into coma ; she recovered after the further withdrawal of a small quantity of cerebrospinal fluid, was apparently normal though somewhat drowsy for four hours, then became cyanosed and died suddenly. Though it was apparent from the condition of the patient that sudden death might have occurred at any time, it cannot

be doubted that the operation contributed to her death. In another case, about three hours after the operation, the patient complained of severe headache and vomited frequently, the temperature rose rapidly, and retention of urine necessitating catheterization occurred. It was only after the use of methods directed towards the temporary relief of increased intracranial tension that this patient's condition returned to normal. In the 392 cases collected by Grant[10] death was directly attributable to ventriculography in 32 cases. Of the 37 cases investigated by ventriculography in Cushing's clinic and referred to by Cairns,[32] 2 died as a direct result of this procedure, and serious symptoms were produced in a third. In one case urgent symptoms appeared one and a half hours after the operation as a result of a great increase in the intracranial pressure, and in another serious symptoms appeared only at the end of three days. Penfield[9] reported a case in which severe headache followed by sudden death occurred thirty-six hours after the operation in a patient in whom the ventriculogram had shown the presence of secondary hydrocephalus. Bingel[8] collected the reports of 6 cases of death following the introduction of air by the spinal route, and of 3 cases in which the ventricular route had been employed. In 7 of these cases there had been ventricular block, in 5 as the result of a tumour above the tentorium, and in 2 owing to a subtentorial tumour. In 2 of Denk's[36] cases included in the above series air was introduced by the lumbar route and death occurred after a long interval.

Penfield[9] has discussed the dangers of ventriculography other than the risk of sudden death or of death at an interval after the operation. In 50 per cent of his cases there occurred a moderate febrile reaction, consisting of a rise of temperature within twenty-four hours and lasting up to four days. Increased intracranial tension with vomiting, drowsiness, and increased headache for as long as one week were present in some cases. Vomiting and rapid coma persisting for one hour supervened in one case in which the lumbar route was employed, and was thought to be due to the collection of air in the subarachnoid space and around the medulla. Penfield mentioned that this type of reaction did not occur in cases in which the ventricular route was employed, but our *Case* 2 provides an example of this reaction under those circumstances. Coma and convulsions occurred in one case and disappeared in four hours after the removal of the air from the ventricles. Frequently, staining of the cerebrospinal fluid with blood was noted towards the end of the operation, probably as the result of a meningeal reaction. This reaction was also mentioned by Hermann[37] and by Mader.[38] The former found 18,000 and 11,000 cells in the fluid at the time of a second puncture, and the latter noted that with successive withdrawals of fluid the percentage of lymphocytes steadily increased. Penfield reported one remarkable case of infection of the ventricles during the operation. Herniation of the cerebellum into the foramen magnum was particularly apt to occur if an increase in intracranial tension was already present; and bulbar paralysis, coming on quickly and disappearing in a short time, was liable to occur especially when the lumbar route was employed. A slow increase in intracranial pressure with vomiting, headache, and drowsiness occurred in some cases in which the ventricular route was employed, but in such cases sudden death might result. Penfield

pointed out that, in the presence of ventricular block, an increase in the intraventricular pressure usually occurred an hour or two hours after the puncture and the introduction of air, and the pressure had been found to be definitely higher at the time of a second puncture than when the injection was just finished. He suggested that this post-operative increase in pressure might be due either to the irritating action of the air or to the sudden reduction of pressure during the operation stimulating an increased production of cerebrospinal fluid. In 20 infants in whom air was injected by the lumbar route Mader[38] observed a constant reaction—the child cried, then became quiet and pale, vomiting and changes in the pulse occurred, and in the more severe cases respiration ceased.

From the consideration of these reactions following ventriculography, it will be obvious that they are sufficiently frequent and severe to suggest that this method of examination should be used only when a definite diagnosis and localization is not possible by other means. The clinical results of diagnosis and localization vary with different individuals and the necessity for ventriculography varies in consequence. It is equally obvious, however, from Grant's figures that it should be used without hesitation in those cases in which the general symptoms are definite, not only to determine the size and position of the ventricles and to determine the presence or absence of a tumour, but also to decide whether exploration or an attempt at surgical removal is likely to meet with success. It definitely increases the proportion of cases of intracranial tumour in which surgical intervention is possible, and at the same time reduces the number of negative explorations. In some quarters, however, ventricular estimation according to the method recommended by Dandy[31] is preferred to ventriculography as being the safer procedure.

Certain procedures have been recommended for dealing with the reactions which are apt to follow ventricular puncture and ventriculography. Of these, the most practicable appear to be the following: (1) The replacement of cerebrospinal fluid by air should be carried out gradually and with only gradual alterations in pressure—that is, the fluid should be withdrawn in very small amounts and replaced with air at each stage. (2) If an unfavourable reaction should occur during the operation, the air should be removed and replaced by Ringer's solution or the previously removed cerebrospinal fluid. As, however, dangerous reactions are liable to occur when a large quantity of fluid is still present, it may be possible only to remove a further small quantity of fluid in an attempt to relieve the symptoms. (3) As the air injected can always occupy a larger volume than the fluid removed, the volume of air injected should always be less than that of the fluid removed. (4) When ventricular block has been shown to be present, it is necessary to anticipate the subsequent rise of pressure within the ventricles by the use of hypertonic saline intravenously, or a saline cathartic by the mouth or rectum. (5) In cases in which ventricular block is suspected the patient may be subjected to decompression immediately the condition is diagnosed and within two hours of the injection of air into the ventricles. The decompression operation may be carried out either as an operation in itself or as the first stage of a subsequent attempt at removal if the case is suitable.

According to Grant, early operation will remove the risk of all the severe after-effects of ventriculography.

The choice of the method to be employed in injecting air for ventriculography in cases of suspected secondary hydrocephalus needs some consideration, and resolves itself into a discussion of the relative advantages and disadvantages of the direct ventricular and of the lumbar route. Of the latter method we have had no experience, but reference to the literature on the subject would appear to point to a very definite choice between the two procedures. According to Penfield,[9] bulbar paralysis, a slow increase in pressure resulting in sudden death, and herniation of the cerebellum into the foramen magnum are more liable to take place when the lumbar route is used. According to Mader,[38] the injection of air by the lumbar route in infants resulted in severe reactions in every case, and in death in one case; whence it was concluded that the spinal route should be used only with great care. Of the nine deaths after ventriculography recorded by Bingel,[8] six occurred after lumbar injection. Denk's[36] three fatal cases were all the result of injection of air by the lumbar route, whereas he had carried out thirty injections by the ventricular route without any serious difficulty. Denk concluded therefore that the injection of air by the lumbar route was never justified in the presence of increased pressure unless preceded by a ventricular decompressive puncture. Jacobaeus[21] apparently favoured the ventricular route in preference to the lumbar route, recognized that it was probably of more value in diagnosis, and drew attention to the possible use of cerebral puncture as a therapeutic agent. Waggoner,[39] basing his opinion on an analysis of ten cases, preferred the spinal route for the purpose of obtaining a roentgengraphic record of the ventricles, cisternæ, and the subarachnoid spaces, and pointed out that it was particularly valuable in the differentiation of the obstructive and communicating types of hydrocephalus.

Opinions differ as to the most suitable site for ventricular puncture. Grant[40] recommends that the site of puncture should be 7 cm. above the occipital protuberance and 1·5 cm. lateral to the middle line, and claims that this site has the following advantages: (1) The site of puncture allows the needle to pass through a silent area of the brain. (2) A puncture at this site permits a direct approach to the vestibule of the lateral ventricle, which is its widest part and that least likely to be collapsed. (3) With the head back, efficient drainage of the ventricles can be obtained. Dandy recommends the occipital route in order to avoid the possibility of hæmorrhage into a cerebral tumour, as a tumour in that region can be ruled out by a careful chart of the visual fields. Others again advise puncture of the inferior horn of the lateral ventricle through an opening 1·5 in. above Reid's base line and the same distance behind the external auditory meatus.

According to the majority of writers, the most important factor for success in ventriculography is the removal of all fluid; but equal stress is laid upon the removal of the fluid and the replacement with air in small amounts at a time, and upon the introduction of a volume of air less than that of the fluid removed. It is essential that antero-posterior, postero-anterior, right lateral, and left lateral skiagrams should be taken, and if possible

stereoscopic skiagrams. Great care must be exercised to keep the head perfectly still while the exposure is being made if a good outline of the ventricular system is to be obtained. In the antero-posterior skiagrams it is essential that the head is kept absolutely in the middle line, otherwise some distortion of one or other ventricles may occur and false deductions be made.

In the interpretation of the ventriculograms much experience of both the normal and abnormal features of the ventricles is needed. Normal variations are not at all uncommon, especially in the lateral ventricles, as has been shown by Penfield.[9]

In general, intracranial tumours produce either symmetrical or asymmetrical variations in the position, shape, and size of the ventricles. The asymmetrical variations are due either to a tumour within the cerebral hemisphere and lateral to the middle line, or to direct impingement of a tumour on the lateral ventricle, and not to obstruction of or interference with the free circulation of the cerebrospinal fluid. Tumours in the region of the foramen of Monro may be partially or completely blocking that opening, and a unilateral hydrocephalus may be produced which can readily be demonstrated in an antero-posterior skiagram. Tumours in the occipital lobe may obliterate the descending horn of the lateral ventricle, evidence of which can be seen in a lateral skiagram, especially if stereoscopic views be taken. In the same way large tumours of the frontal lobe may exert enough pressure almost to obliterate the descending horn of the lateral ventricle, and so produce an irregularity in the outline of a lateral skiagram. As a general rule it may be said that asymmetry of the lateral ventricles is strong evidence of a cerebral tumour on the side of the small ventricle.

Excepting for the rare cases in which a tumour of the midbrain obstructs the outflow of cerebrospinal fluid from one lateral ventricle to a less extent than from the other, obstruction of the free circulation of the cerebrospinal fluid usually results in a more or less symmetrical dilatation of the two lateral ventricles, and may be produced by a tumour in the mid-line between the two hemispheres, by one involving the foramen of Monro or the third ventricle, or by one blocking the outflow of fluid in the aqueduct of Sylvius, the fourth ventricle, or the cistern. If it be accepted that a subtentorial decompression is essential in the case of subtentorial tumours—a question which will be discussed at a later stage—it is necessary to differentiate carefully between a supratentorial and a subtentorial tumour in the region of the third ventricle. It has been suggested that two useful points of differentiation are the following: (1) If the third ventricle can be seen, the block is below the tentorium; and (2) A subtentorial tumour to one side of the middle line may impinge upon the posterior horn of the lateral ventricle and distort it. Elsberg and Silbert[41] have added the following interesting point of differentiation: that a block above the tentorium distends the anterior and inferior horns of the lateral ventricle equally, whereas one below the tentorium distends the former more than the latter.

Generally it is found that the antero-posterior and postero-anterior views are the most valuable, but in arriving at a conclusion on the ventriculograms it is essential to consider only the defects which are present in all the views and to correlate the results with the clinical findings.

In some cases there can be no doubt that ventricular estimation may take the place of ventriculography, and it is a safer procedure. In this operation the lateral ventricles are tapped and the amount of fluid withdrawn from each is noted. Any amount over 30 c.c. points to a condition of hydrocephalus, and if the amounts from the two ventricles are not the same it is strong evidence of a tumour above the tentorium.

5. OBSERVATIONS AT THE TIME OF OPERATION.

Observations made at the time of operation, and especially in the course of operation for mobilization of the tentorium, are useful in confirming a diagnosis of secondary hydrocephalus. The cerebral hemispheres are found to be tense and firm to palpation, the gyri are flattened, and the superficial veins are distended. If the pressure is high, the dura and the brain tissue are forced into the wound on removing the bone-flap. Distension on one side more than on the other may help to localize the cause of the obstruction in the midbrain or between the two cerebral hemispheres, obstructing the circulation of fluid from one lateral ventricle more than from the other; while a collapse of the ventricle on one side associated with dilatation on the other side suggests a tumour involving the cerebral hemisphere on the collapsed side. Equal distension of the two ventricles suggests that a midbrain tumour has obstructed both foramina of Monro, or that the tumour causing the block is at a lower level.

In addition, observations of the tenseness of the cerebral hemispheres during lumbar puncture after the bone-flap has been removed may show that no decrease in pressure occurs with the removal of fluid from the spinal canal, and confirm the opinion that a block exists in the hindbrain.

IX. THE TREATMENT OF SECONDARY HYDROCEPHALUS.

The effect of the presence of secondary hydrocephalus upon the treatment to be employed would appear to be the following. Measures designed to facilitate the absorption of cerebrospinal fluid, such as the intravenous injection of hypertonic saline solution or the use of magnesium sulphate by the mouth or the rectum, may be necessary as a preliminary step. We must state, however, that we have never seen any prolonged benefit from repeated enemata of concentrated magnesium sulphate, nor from the use of hypertonic saline intravenously. These measures have been used successfully to carry a patient over the danger period, such as that due to the sudden increase in intracranial pressure after ventriculography.

Following such preliminary urgent treatment, surgical methods of relieving the increased intracranial tension are necessary. Repeated ventricular puncture may be considered, but it would appear that this method is likely to be of value only in those cases in which time is necessary for a local inflammatory or obstructive lesion to recover. It was used successfully in a case reported by Gordon.[30]

When surgical decompression is considered, it has to be decided whether decompression above or below the tentorium should be employed, even when there are some indications that the cause of the blockage is in the posterior

fossa of the skull. Each case has to be considered on its merits, but the balance of evidence would appear to be in favour of a supratentorial decompression in association with exploration of the posterior fossa. The general principle would appear to be to attack general symptoms above the tentorium and only definite localizing symptoms below the tentorium, and, when necessary, to combine both objects in one operation.

The treatment of secondary hydrocephalus is, therefore, the removal of the tumour wherever possible, but this is rarely possible, more particularly when the tumour cannot be localized. The treatment where the tumour cannot be removed is simply that of decompression. The intermusculo-temporal decompression gives quite good results and considerable temporary relief. For cases in which the tumour is subtentorial the best results are obtained by turning down an osteoplastic flap, and exposing the occipital lobes and the cerebellar hemispheres by means of Souttar's craniotome. The constriction of the tentorium at once becomes loose, as the tentorium no longer has its bony attachment behind the occipital bone.

X. SUMMARY AND CONCLUSIONS.

1. The influence of secondary hydrocephalus upon the diagnosis and localization of intracranial tumours is discussed, and the methods of investigation and treatment are detailed.

2. The different views propounded to explain the occurrence of hydrocephalus in the course of intracranial tumours are reviewed.

3. Seven cases of intracranial tumour complicated by secondary hydrocephalus are described to illustrate the difficulties in diagnosis and localization in the presence of secondary hydrocephalus.

4. The differential diagnosis of intracranial tumour complicated by secondary hydrocephalus is discussed.

5. Secondary hydrocephalus as a source of confusion in localization is considered; attention is drawn to the false localizing signs which may appear as a result of that condition; and stress is laid upon the chronological order of the physical signs.

6. The investigation of a case of secondary hydrocephalus is discussed; the clinical features of the condition are described, and the value of puncture methods, X-ray examination, ventriculography, and observations at the time of operation are considered.

7. Ventriculography should be employed in the investigation of a case only when all other methods of examination have failed to establish a diagnosis and to localize the lesion; and, in carrying out this procedure, ventricular puncture is to be preferred to spinal puncture.

8. In view of the questions raised in this paper, the treatment of secondary hydrocephalus should be instituted as early as possible commensurate with thorough investigation of the patient. It should provide for the relief of pressure above the tentorium even when the local lesion is situated in the posterior fossa of the skull—a result which is best achieved, when symptoms of a subtentorial lesion are present, by the operation for mobilization of the tentorium, as has been amply illustrated by some of Souttar's cases.

We are indebted to our colleagues on the staff of King's College Hospital and of the West End Hospital for Diseases of the Nervous System for permission to include cases under their care, and especially to Dr. W. E. Carnegie Dickson for detailed pathological reports on four of the cases described.

REFERENCES.

[1] FAIVRE, *Comptes rend. de l'Acad. des Sci.*, 1854, xxxiv, 424 (quoted by Greenfield and Carmichael in *The Cerebrospinal Fluid in Clinical Diagnosis*, 1925).
[2] DANDY, W. E., *Ann. of Surg.*, 1919, lxx, 129.
[3] WEED, L. H., *Jour. Med. Research*, 1914–5, xxxi, 21.
[4] STOPFORD, J. S. B., *Brain*, 1929, li, 485.
[5] SARGENT, P., *Jour. Anat. and Physiol.*, 1910–1, xiv, 69.
[6] BRAIN, W. R., *Brain*, 1925, xlviii, 105.
[7] SOUTTAR, H. S., *Brit. Med. Jour.*, 1929, i, 53.
[8] BINGEL, A., *Med. Klinik*, 1923, xix, 637.
[9] PENFIELD, W. G., *Arch. of Neurol. and Psychiat.*, 1925, xiii, 580.
[10] GRANT, F. G., *Ibid.*, xiv, 513.
[11] DANDY, W. E., and BLACKFAN, D. B., *Amer. Jour. Dis. Child.*, 1914, viii, 406.
[12] DANDY, W. E., and BLACKFAN, D. B., *Ibid.*, 1917, xiv, 424.
[13] DANDY, W. E., *Johns Hopkins Hosp. Bull.*, 1921, xxxii, 67.
[14] DANDY, W. E., *Ann. of Surg.*, 1925, lxxxii, 199.
[15] YOUNG, A. W., *Canad. Med. Assoc. Jour.*, 1926, N.S. xvi, 952.
[16] BRAMWELL, B., *Brain*, 1899, xxii, 66.
[17] BRAMWELL, B., *Brit. Med. Jour.*, 1898, ii, 964.
[18] SPILLER, W. G., *Amer. Jour. Med. Sci.*, 1902, cxxiv, 44.
[19] OPPENHEIM, *Monats. f. Psychiat. u. Neurol.*, 1905, xviii, 221.
[20] RHEIN, J. H. W., *Jour. Amer. Med. Assoc.*, 1918, lx, 1933.
[21] JACOBAEUS, H. C., *Acta Med. Scand.*, 1923, lix, 666.
[22] CUSHING, H., *The Pituitary Body and its Disorders*, 1912, 201.
[23] MARIENESCO, and GOLDSTEIN, M., *Nouv. Icon. de la Salpêtrière*, 1909, xxiii, 628.
[24] GOLDSTEIN, KURT, *Arch. f. Psychiat. u. Nervenkrank.*, 1910, lxiii, 126.
[25] STUMPF, *Virchow's Arch.*, 1912, ccix, 399.
[26] POLLOCK, L. J., *Jour. Amer. Med. Assoc.*, 1915, liv, 395.
[27] STRAUCH, A., *Ibid.*, 1919, lxii, 1731.
[28] SCHULTZ, *Deut. med. Woch.*, 1923, xlix, 1072.
[29] LAIGNEL-LAVASTINE, and CL. VINCENT, *Rev. neurol.*, 1929, xxxvi, 106.
[30] GORDON, A., *Jour. Amer. Med. Assoc.*, 1927, lxxviii, 1234.
[31] DANDY, W. E., *Surg. Gynecol. and Obst.*, 1923, xxxvi, 641.
[32] CAIRNS, H., *A Study of Intracranial Surgery*, 1929.
[33] MCKENZIE, K. G., and SOSMAN, M. C., *Amer. Jour. Roentgenol.*, 1924, xi, 171.
[34] SOSMAN, M. C., and PUTNAM, T. J., *Ibid.*, 1925, xiii, 1.
[35] VAN DESSEL, A., *Arch. franco-belge de Chir.*, 1925, xxviii, No. 10 (quoted by Cairns).
[36] DENK, W., *Zentralb. f. Chir.*, 1923, xii, 471.
[37] HERMANN, G., *Med. Klinik*, 1922, xviii, 1146.
[38] MADER, A., *Ibid.*, 1923, xix, 1427.
[39] WAGGONER, R. W., *Amer. Jour. Med. Sci.*, 1927, clxxiv, 459.
[40] GRANT, F. G., *Radiology*, 1927, ix, 388.
[41] ELSBERG, A., and SILBERT, S., *Arch. of Neurol. and Psychiat.*, 1925, xiv, 489.

VOLKMANN'S ISCHÆMIC CONTRACTURE TREATED BY TRANSPLANTATION OF THE INTERNAL EPICONDYLE.

By NORMAN HODGSON,

ASSISTANT SURGEON TO THE ROYAL VICTORIA INFIRMARY, NEWCASTLE-UPON-TYNE.

In the British Journal of Surgery, October, 1928, Hamilton Bailey recorded a case of Volkmann's ischæmic contracture treated by the transplantation of the internal epicondyle of the humerus. Stimulated by this

Fig. 171.

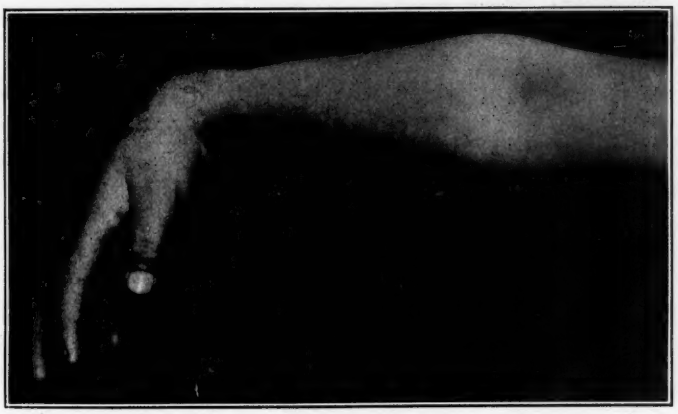


Fig. 172

article I determined to try the operation in a case which had hitherto resisted all efforts to overcome the contracture, and the result has far exceeded my expectations.

The case was one of a girl, age 14, who some eight years previously had sustained a fracture of the lower end of the right humerus. The method by which the fracture was treated could not be ascertained. When first seen four years ago she had a typical Volkmann's contracture. Gradual extension of the wrist, massage, and movement were tried over a long period, but the improvement made was negligible.

Figs. 171 and 172, made before the operation, show the flexion of the fingers when an attempt was made to extend the wrist, and that the fingers could only be extended by fully flexing the wrist.

FIG. 173.

In November, 1928, I carried out the operation on the lines described by Hamilton Bailey. My experience follows his exactly; there was no dramatic relief of the contracture, but at the end of three weeks there was an appreciable improvement in the amount of extension obtainable. Massage and movement are being continued, together with gradual extension of the wrist on a 'cock-up' splint.

Fig. 173 shows the condition of the hand on Jan. 31, 1929. The patient has now good use of the fingers and wrist, and what was previously practically a useless member is now a useful limb. I hope that in the course of time still further extension of the wrist will be possible.

A FURTHER NOTE ON KÜMMELL'S DISEASE.

By J. CARDIS, G. F. WALKER, and R. H. OLVER,
GENERAL INFIRMARY, LEEDS.

As a sequel to our previous article on Kümmell's disease[1] we wish to place on record the pathological findings subsequently ascertained in one of the cases therein recorded. The present photograph (*Fig.* 174) refers to *Case* 7 in our previous account.

Fig. 174.

In March, 1925, he was kneeling, and was struck on the back by a falling stone. He was kept in bed for several weeks on account of pain and shock. X-ray examination at this time revealed no bony spinal lesion at the site of the injury. In the summer of 1925 he was up and about, much relieved, but still complaining of residual soreness. He could walk about fairly comfortably. In October he complained of a great exacerbation of the dorsal pain. His back became very painful when he walked. Examination revealed several healed abrasions on the back and a kyphosis at the dorsilumbar junction. There was considerable local tenderness with much pain and limitation of movement at the level of the 1st lumbar vertebra. X-ray examination showed that this vertebral body was collapsed and had assumed a wedge shape typical of Kümmell's disease. The patient was thereupon fitted with a jacket giving spinal support. His symptoms regressed and his spinal lesion became stationary. In April, 1926, insanity, not relevant to Kümmell's disease, supervened and he was certified and removed to an asylum. Here his spinal lesion remained stationary for two years, and finally the spinal jacket became quite unnecessary, though the patient would never part with it.

Sequel.—In September, 1928, the patient died, and by the courtesy of Professor Shaw Bolton his dorsilumbar spine was added to the collection of similar specimens in the Pathological Museum of the Leeds School of Medicine. The photograph (*Fig.* 174), which we think calls for no comment, shows the typical wedge-shape deformity and collapse of the 1st lumbar vertebra.

Recent authoritative literature,[2] notably from the original pen, has stabilized Kümmell's disease as a clinical entity. We do not propose to recapitulate our former article, but we would like to emphasize in the above case the characteristic salient features of the malady. The full sequence is: (1) An injury, direct or indirect, grave or trifling, to a vertebral body; (2) A

period of disability according to the gravity of the trauma; (3) A latent period; (4) A recurrence of local pain and severe disability due to (5) A crumpling collapse of a selected vertebral body, and its assumption of a wedge shape as seen in sagittal section; (6) The arrest of the process by orthopædic support.

We are again indebted to Professor Maxwell Telling for permission to record this case.

REFERENCES.

[1] "Kümmell's Disease", *Brit. Jour. Surg.*, 1928, xv, 616.
[2] KÜMMELL, "Die posttraumatische Wirbelerkrankung sogenannte Kümmellsche Krankheit", *Monats. f. Unfall.*, 1928, xxxv, 65.
HEILIGTAG, "Verwechslung zwischen Spondylitis tuberculosa und Kümmellsche Krankheit bei der Unfallbegutachtung", *Ibid.*, 1927, xxxiv, 162.
BRANDIS, "Wirbelsaulenerkrankung (Kümmellsche Krankheit) als Unfallfolge", *Med. Klinik*, 1927, xxxii, 1228.
JONES, F., *Jour. Amer. Med. Assoc.*, 1923, lxxxi, 1860.

ON THE TRANSFUSION OF CITRATED OR DEFIBRINATED BLOOD THROUGH A FINE NEEDLE.

By H. MOLL,

MEDICAL TUTOR IN THE UNIVERSITY OF LEEDS.

Blood transfusion has developed from an emergency operation, confined to the operating theatre, into a routine mode of medication, which often has to be repeated at frequent intervals and be performed either in the ward of a hospital or at the home of the patient. Although many methods have been devised during the last ten years, there is yet a demand for a simple and reliable procedure which can be carried out at the bedside, without much assistance, with minimum discomfort both to the donor and to the recipient, and with success.

One of the first difficulties in performing a blood transfusion is to find a suitable donor. Now that the glamour of giving one's blood to a patient has largely died out, owing to the fact that blood transfusion is often used not as a desperate effort to save life but as a remedial measure, voluntary donors who are not related to the patient, and even professional donors, demand that no incision be made which needs sutures and subsequent dressings and which will leave a scar on the arm. The use of a needle, even of large bore, on the other hand, has proved to be unsatisfactory in many cases, as the flow of blood is never as good as when using a cannula; the flow may slow down after the first 200 to 300 c.c. and the blood may clot inside the needle, thus preventing any further collection of blood unless another vein is tapped with a fresh needle.

Dr. Herbert French has devised a 'needle' which is a cross between a cannula and an ordinary needle, having a point with three facets like a bayonet and a stem made to a conical shape so that the bore at the base is considerably greater than at the point. The needle is so sharp that it will pierce the skin like butter, it is easily introduced into a vein, and it ensures a splendid flow of blood, without risk of clotting. French's needle (*Fig.* 175 B) is connected directly to the jar for collecting blood (A) by thick rubber tubing which cannot kink, and there are no metal or glass connections between to cause friction or to hold up the flow of blood along its course, as the rubber tubing is threaded straight through the rubber bung of the jar. The blood flows by gravity, and it is neither necessary nor advisable to produce a vacuum inside the jar, as this tends to hinder the flow by collapsing the vein. There is no risk of contaminating the blood by holding the jar below the donor's arm, as the jar is closed by a rubber bung. A pint or more of blood is thus quickly drawn off, and on withdrawing the needle the small triangular wound like a leech bite soon closes and stops bleeding on lifting the arm above the head for a few minutes. There is no incision, no injury

to the vein, and the same vein may be used repeatedly for subsequent transfusions. It is not advisable, however, to use a donor more than twice for the same patient, as the recipient becomes as it were sensitized to the blood.

The use of sodium citrate as an anticoagulant is the easiest way of transfusing blood, although it is not always harmless. Mellon[1] has shown the variability of the chemical reaction of commercial sodium citrate, and there is probably also an individual variation in the tolerance of different people.

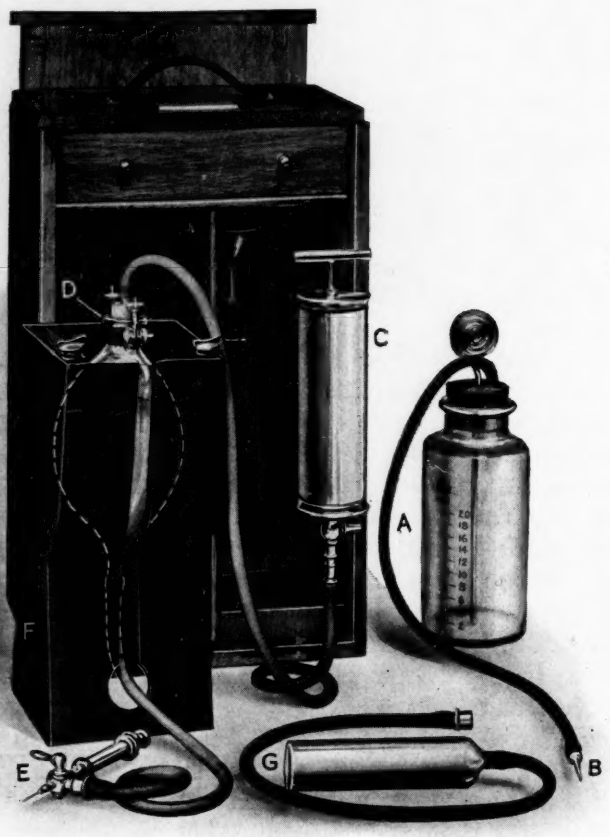


FIG. 175.—Diagram of the apparatus. A, Graduated jar for collecting blood. The graduations are both in ounces and cubic centimetres. The curved rod is used for defibrinating the blood; B, French's needle; C, Pump; D, Reservoir containing blood after having been citrated or defibrinated and filtered; E, Two-way stop-cock with hypodermic syringe, needle, and tube from reservoir, all attached in readiness for use; F, Heating stand with two hot-water tanks on either side of the reservoir. The reservoir may be taken out of the stand; G, Saline infusor.

Lederer[2] and others claim that the addition of sodium citrate increases the incidence of reactions. This drug, however, is not the only factor in producing a post-transfusion reaction. The reaction may in some cases be anaphylactic, due to some sensitive protein in the donor's blood, or it may be caused by slight incompatibility, especially when stock grouping rather than direct testing of the donor's and recipient's blood is relied upon; lastly

it may be due to incipient coagulative changes if the blood is not drawn off quickly enough and well filtered. Keynes,[3] while admitting the possible influence of citrated transfusions in causing reactions, regards their occurrence as of little importance and as greatly overweighed by the advantages of the method.

In view of these objections which may be made to the citrate method, the collecting jar (*Fig.* 175 A) in my apparatus has been adapted also for the defibrination method. When using the defibrination method, Skinner[4] has recommended the use of a special curved rod, which is inserted into the rubber bung with its curled end pointing downwards and touching the bottom of the jar. The jar is kept in a continuous rotary motion during the whole time the blood is being collected and for at least five minutes afterwards. Fibrin will collect in a lump on the curled end of the rod and can then be removed.

If the citrate method is used, the required amount of a freshly prepared and sterile solution of sodium citrate is poured into the collecting jar, and the central rod is inserted with its curled end pointing upwards. Opinions differ as to the strength of the solution to be used. I use the smallest amount of citrate sufficient to prevent clotting, or 10 c.c. of a 2 per cent solution to every 90 c.c. of blood, although some writers favour larger doses. Robertson[5] recommends 160 c.c. of a 3·8 per cent solution for 750 c.c. of blood as it gives an isotonic solution. Spriggs[6] uses a 4 per cent solution, of which 100 c.c. are added to each 500 c.c. of blood.

The citrated or defibrinated blood is decanted from the collecting jar (A) into the reservoir (D) and at the same time it is strained through two layers of gauze or cheese cloth. Prior to pouring the blood into the reservoir a small amount of normal saline is made to flow through the rubber tubing, the two-way stop-cock (*Fig.* 175 E and *Fig.* 176) and needle, in order to expel any air contained therein.

The reservoir (D) is placed in a heating stand (F) provided with two water tanks on either side to keep the blood warm. A chief difficulty in performing a blood transfusion is to deliver the blood to the recipient. The cannula method entails dissecting a vein to the discomfort of the patient, while if a needle of moderate bore is used, as suggested by methods described heretofore, there is always some anxiety in introducing the needle into the vein when the vein is small or contracted. In gravely exsanguinated or nervous patients the vein may be spastically contracted so as to make it impossible for the blood to flow into the vein after the needle has been inserted. In order to overcome these difficulties, I have introduced the use of very fine intravenous needles (size 21 Standard Wire Gauge), and this has been made possible by a specially devised pear-shaped container (D), a strong pump (C), and a two-way stop-cock (E). The pear-shaped container is connected to a pump, and the mouth of the container is closed by a rubber stopper which can be firmly fixed by two screw clips. By using a reservoir of this shape, practically all the blood can be pumped out under great pressure without any risk of blowing air into the vein. The pump is made to the same design as that used in a Potain's aspirator, but is three times larger and will enable blood to be pumped through a very fine needle.

Lastly a two-way stop-cock (*Fig.* 175 E and *Fig.* 176) has been devised which so simplifies the technique of delivering the blood to the recipient that this may be done as easily and with as little discomfort to the patient as any ordinary intravenous injection. To it an ordinary hypodermic syringe, a needle, and the tubing leading from the reservoir are attached. The tap of the stop-cock can be turned in such a way as to have either the syringe and needle communicating (*Fig.* 176 *a*), or the syringe cut off and the needle communicating with the tube from the reservoir (*Fig.* 176 *b*). With the tap turned to *a*, the operator introduces the needle into the vein, draws some of the recipient's blood into the syringe to ensure that the needle has been correctly inserted, thereupon turns the tap round to *b*, and rapidly connects the needle to the reservoir. In this way one eliminates the tedious procedure of fitting the tube on to the needle after the vein has been punctured, which often entails spilling of blood and a risk of air embolism, and the whole operation of inserting the needle and connecting it with the transfusion set is made perfectly easy and fool-proof. When the reservoir is nearly empty the tap of the stop-cock is turned back to *a*, thus shutting off the reservoir, and the needle is withdrawn from the vein. The small amount of blood left in the stem of the reservoir and in the rubber tubing is a sufficient safeguard against air being injected into the vein. It is not possible to overlook the fact that the reservoir has been emptied, as the heating stand has a slit-window exposing the whole length of the reservoir, and the flow of blood can be easily watched.

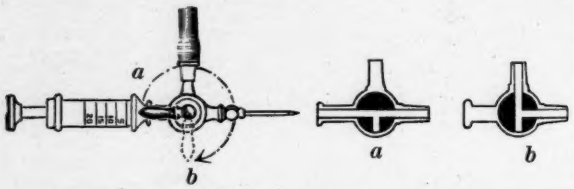


FIG. 176.—Diagram of the two-way stop-cock. *a*. The needle and syringe are communicating. *b*. The tube leading from the reservoir (*Fig.* 175 E) is now connected with the needle.

TECHNIQUE.

To Collect the Blood.—Pour the required amount of sterile 2 per cent solution of sodium citrate into the collecting jar. Place a sphygmomanometer armlet round the donor's arm, raise the pressure to 80, and instruct the donor to clasp and unclasp his fist alternately. With a quick jab introduce French's needle into a vein (median basilic or median cephalic) and allow the blood to flow into the jar by gravity.

When using the defibrination method insert the metal rod with its spiral end pointing downwards touching the bottom of the jar, and do not add any citrate. During the whole time the blood is being collected the bottle is kept constantly revolving gently, thereby swinging the blood content against the spiral. As soon as sufficient blood is obtained the needle is withdrawn and the bottle continuously rotated for about six minutes, at the end of which time the cork is removed; a large single clot of fibrin will be found adhering to the spiral. Before pouring the citrated or defibrinated blood into the reservoir precede with some sterile saline to expel any air in the rubber connections, and while decanting the blood from one jar to the other strain the blood through gauze or cheese cloth. Place the reservoir in the heating stand to keep the blood warm.

To Deliver the Blood.—Place a bandage round the recipient's arm so that it can be easily loosened as soon as the vein has been punctured. With the tap of the stop-cock turned to *a*, introduce the needle into a vein and draw some blood into the hypodermic syringe; if blood flows freely into the syringe it is sufficient proof that the needle has been correctly inserted. Turn the tap to *b*, thereby connecting the needle to the reservoir, loosen the bandage, start to pump, and blood will begin to flow into the vein. When the reservoir is nearly empty turn the tap back to *a* and withdraw the needle from the vein. It takes ten to twenty minutes to deliver a pint of blood, but if necessary the blood can be injected much more slowly.

The apparatus has been thoroughly tested in a large number of blood transfusions over a period of three years and can be strongly recommended. The technique is extremely simple and does not require any operative skill. The apparatus can be easily cleaned and sterilized and the whole set is obtained in a convenient box for carrying it. The transfusion set includes also a saline infusor composed of a cylindrical glass funnel with rubber tubing (*Fig.* 175 G) which can be connected to the two-way stop-cock and needle. The same procedure is followed as described for delivering blood, except that saline is poured into the funnel and allowed to flow into the vein by gravity. The saline infusor may be used for intravenous injection of normal saline, glucose, etc. The whole of my own apparatus, as described above, may be obtained from Messrs. Reynolds & Branson Ltd., of Leeds.

SUMMARY.

Difficulties arising from other methods of blood transfusion, described heretofore, afford a sufficient excuse for devising yet another apparatus having the following advantages:—

1. A very fine intravenous needle (size 21, I.S.W.G.) can be used for delivering blood to the recipient.
2. The operation of inserting the needle into the vein is simplified by a two-way stop-cock enabling the operator to ensure that the needle is correctly placed inside the vein and rapidly to connect the needle with the transfusion set by the mere turn of a tap.
3. There is no risk of clotting.
4. Either the citrate or the defibrination method may be used.
5. Blood transfusion can be carried out at the bedside, as there is very little need for asepsis.
6. Blood is kept warm in a heating stand.
7. The technique is extremely simple, does not require any operative skill, and ensures success every time.

REFERENCES.

[1] Mellon, quoted by Lederer.
[2] Lederer, M., *Surg. Gynecol. and Obst.*, 1923, xxxvii, 221.
[3] Keynes, G., *Blood Transfusion*, Oxford Medical Publications, 1922, 123.
[4] Skinner, E. F., *Brit. Med. Jour.*, 1925, ii, 516.
[5] Robertson, O., *Ibid.*, 1918, i, 477.
[6] Spriggs, E. I., *Ibid.*, 1926, ii, 978.

THE RADIUM PROBLEM.

I. INTRODUCTORY.

By Professor G. E. GASK, C.M.G., London.

The interest of the world of medicine and of the public at large has been stirred by the advances made in the last few years in the treatment of malignant disease by radium. The subject is of such pressing national importance that in April of this year the Radium Sub-Committee of the Committee of Civil Research issued a report on radium in the treatment of cancer, and the Government promised to make a grant of £1 for every £1 subscribed by the public for the establishment of a National Radium Fund, and this now amounts to a large sum. The ready response of the public to this appeal shows its practical interest, and it has been decided that the public contribution shall form part of the Thank-offering Fund for the King's convalescence. Subsequently two bodies were founded to deal with the fund—namely, the National Radium Trust and the National Radium Commission. As the result of this National Fund it is hoped that a large supply of radium will be forthcoming for the use of the medical profession in its campaign against cancer. It is well, therefore, that a survey should be made to consider the present position of radium in the treatment of malignant disease in order to assess its proper value, without either taking an exaggerated view of its therapeutic properties or underrating them.

It has been known for many years that radium has a powerful effect on various affections of the skin, and that rodent ulcer and superficial epitheliomata can often be completely and permanently cured by its use in surface applicators. During the last few years the technique of radium therapy has been elaborated and greatly improved by the use of radium needles, or of seeds containing radium emanation or radon, which are buried in the tissues in or around the growth. Most of the work done in this country has been directed towards the treatment of cancer of the cervix uteri, the rectum, the breast, and the tongue and buccal cavity. These areas have been selected by the various Research Centres, because in these situations growths are easily accessible and the effects of the radium can be seen and judged, whereas in internal cancers it is much more difficult to watch the effect of treatment. It can be stated definitely that in the above-mentioned areas, as the result of long and hard work, of many trials and many errors, a big step forward has been made. In many cases carcinomata of the cervix, breast, rectum, and mouth have entirely disappeared, and the patients remain well and apparently free from disease for several years.

It is astounding—indeed, almost miraculous—to observe the manner in which in a successful case a malignant growth fades and disappears. In one instance a well-marked epitheliomatous ulcer of the tongue disappeared and skinned over in the space of three weeks from the commencement of treatment. The nearest comparison that can be made is the manner in which a gummatous ulcer on the skin heals in a few weeks under appropriate treatment. This makes one wonder what is the action of radium on the growth. Does it kill the cancer cells? One has seen the effect on the cells in Canti's film. Or does it kill the virus or active agent of cancer? There is as yet no answer to this intriguing question.

Remarkable as are the results already obtained, we must not allow ourselves to run away with the idea that radium is the cure for all cancers, for the problem is not an easy one. The attack on the primary growth is, in a sense, the easiest part of the task, for if the growth is accessible, if the radium is implanted in the right place, if the whole of the tumour is irradiated, if the dose is correct, and if the exposure is rightly timed, then one may expect the shrinkage and disappearance of the tumour. In the course of the work one point that has come out quite clearly is that destruction of a primary growth, followed by its complete disappearance, does not in any way affect the growth and extension of metastases, if these are already formed, any more than excision of a carcinoma of the breast by the knife will cure a patient if there are secondary deposits in the viscera. Radium therapy, if it is to be successful, must aim at destroying not only the primary growth, but also any extensions which may have formed in the neighbouring lymphatic glands. We must emphasize again the fact that treatment of cancer, if it is to be successful, whether it takes the form of surgical excision, the cautery, radium, or X rays, must be undertaken early, before dissemination has occurred; and it must be reiterated that an essential part of the campaign against cancer consists in the education and intelligent co-operation of the public so that early diagnosis and early treatment may be secured. Already something has been done in the direction of irradiating the lymphatic drainage area at the same time as the primary growth, as will be seen in the succeeding articles, and the prospects of improving this line of treatment appear to be good.

If one turns now to consider what is the relative value of radium therapy in the treatment of cancer as compared with surgical excision, it will be found a difficult point to assess, for the reason that there are not yet sufficient facts available. In the first place most of the available data concern cancer only of the areas of the body above mentioned, and secondly we have not yet a sufficient number of cases, nor has sufficient time elapsed, to prepare statistics which will show a true picture. The great point in favour of the use of radium is that the effect can be obtained without the extensive and mutilating operations which are at the same time so distressing to the patient and to the surgeon. The operation of introducing the radium is in itself a comparatively slight one, not fraught with any immediate danger, and while the radium is in position the patient suffers little or no discomfort. Under these conditions, and with bright hopes of cure without mutilation, patients are far more ready to consult their doctors with hope of relief, instead of hiding their tumours till it is too late for any form of radical treatment.

Against the use of radium, and in favour of surgical excision, must be placed the danger of leaving a portion of a cancerous growth non-irradiated, and of leaving infected lymphatic glands which might be removed by excision. The difficulty of arriving at the proper dose and exposure must also be reckoned. On the one hand the radiation may be too little; on the other hand it may be too much, resulting in burning or sloughing of tissues and the formation of obstinate and slow-healing ulcers. Last, but not least, there is the danger to the health of workers in radium. Radium is a double-edged weapon, as the pioneers in this form of therapy have found to their cost. The housing, staffing, and maintenance of a radium institute for surgical purposes require very careful management and supervision, and are therefore costly.

The above seems to be a long array of arguments against the use of radium, but a little consideration will show that most of the points raised may be overcome by care and forethought.

Most of the results achieved in this country have been by means of superficial applications, by buried needles, by radon seeds, or by the combination of these three methods. Little has been done in the radiation of tumours from a distance by large quantities of radium—that is, 4 or more grm. at a time. This is known as distance radium therapy, or more commonly as the 'bomb' treatment, because a large quantity of radium is placed in a container —the 'bomb'—and applied in such a way that the radium rays may penetrate the affected area. We have not had sufficient radium to do much in this way, but it is a method which urgently requires investigation.

The summary of the argument is that a good case has been made out for the employment of radium. Hope for the cure of cancer is bright. A new weapon and a powerful weapon has been placed in the hands of the medical profession, though just how good it is impossible to say as yet. Nor is it yet possible to say whether patients with malignant disease should be treated with radium alone, or with radium combined with surgery or with X rays. One would think it probable that a judicious use of them all may be required; but at any rate it seems clear that clinicians must be familiar with a use of all three methods.

What is required at the moment is work, intensive work, on the many problems which present themselves, combined with careful documentation of the methods used and registration of the results obtained. This calls for the co-operation of physicists, clinicians, radiologists, pathologists, and research workers. Some brilliant work has already been done, but much more requires to be accomplished before the story of radium can be told.

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF FIBROSARCOMA OF THE CERVICAL MENINGES.

By CECIL P. G. WAKELEY,
SURGEON TO KING'S COLLEGE HOSPITAL, LONDON.

Intradural tumours of the cervical meninges are comparatively rare, and although such cases are seen and operated upon successfully, very few have been reported in this country. The following case is of interest as immediate relief followed operation, although the symptoms were of two years' standing.

History.—L. P., age 28, was admitted to King's College Hospital in May, 1923, under the care of Dr. Aldren Turner, complaining of unsteady gait and pains in his shoulder. The onset of his complaint took place two years before admission to hospital, when the patient noticed pain in his right forearm. This was at times so severe as to prevent him from sleeping, and it gradually extended from the forearm to the shoulder. He stated that any sudden movement of his neck caused him severe pain, and to prevent this he kept his neck constantly bent downwards towards his chest.

About one year prior to admission to hospital he noticed weakness in his right leg. This was thought to be of a rheumatic nature, for which his doctor prescribed a liniment. However, the weakness became more marked, and the patient was unable to walk without the aid of a stick. When walking he could only drag the right leg along, and found it impossible to lift it from the ground. In January, 1923, he contracted influenza, and was in bed for eight weeks. He had lost two stone in weight since the commencement of his illness. When he was able to get about again in March, 1923, he found it very difficult to walk, even with the assistance of two sticks. He also complained of weakness in the right hand.

On Examination.—When seen in May, 1923, he was found to be a fairly well nourished man. The right arm was considerably thinner than the left, although he was right-handed at his work as a joiner. There was a difference of two inches in the circumference of the upper arm on the two sides. There was very little difference in the measurements of the lower extremities, although the right leg was slightly smaller than the left. There were no motor disturbances on the left side of the body, while on the right side there was a well-marked atrophy of the muscles of the thenar and hypothenar eminences. The interossei appeared to be completely paralysed, as the patient could not separate his fingers at all. There was definite weakness of the flexors and extensors of the fingers. He could only stand with difficulty, and always fell

forwards and towards the right when his eyes were closed. Nothing abnormal was discovered in the chest or abdomen. The pulse-rate was 80, the respirations were 18, and the blood-pressure was 140 in the brachial artery.

There was no paralysis of any of the cranial nerves. The pupils reacted well both to accommodation and light. The right pupil was thought to be slightly smaller than the left; the fundi appeared normal. The knee- and ankle-jerks were increased on both sides, ankle clonus was very marked on

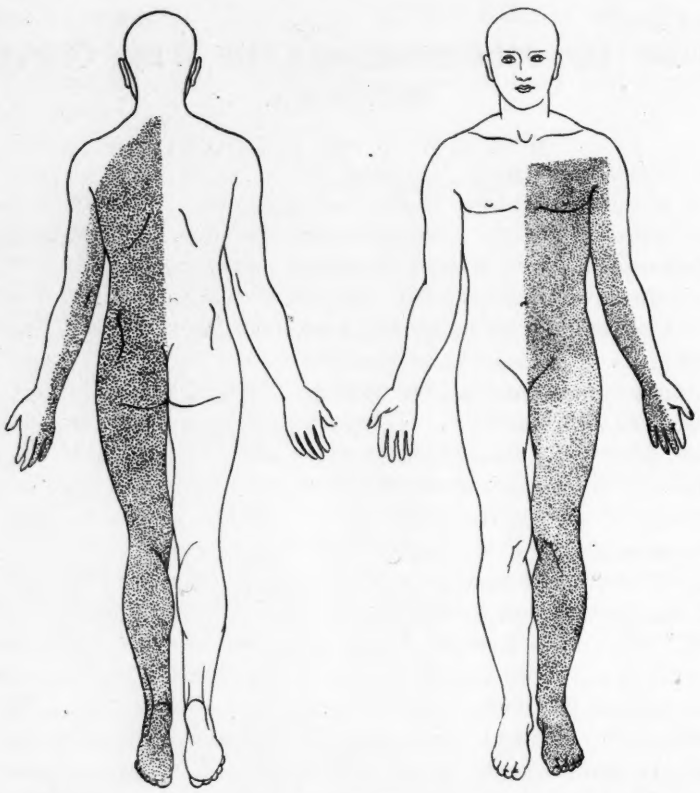


FIG. 177.—Showing areas of loss of perception to heat and pain.

the right side. The biceps- and triceps-jerks were increased in the right arm; the abdominal reflexes were absent. With regard to sensory disturbance, there was no interference with the transmission of tactile impressions from either side of the body, but perception of heat and pain was lost over the whole of the left side from the level of the second intercostal space (*Fig.* 177). There was no loss of muscle sense in the extremities.

Skiagrams of the cervical spine did not demonstrate any abnormality in the bones.

FIG. 178.

OPERATION.—Laminectomy was performed on May 10, under intratracheal ether anæsthesia. An incision was made as shown in *Fig.* 178, extending over the lower four cervical and upper two thoracic spinous processes. The spinous processes were bared of muscles and ligamentum nuchæ. The muscles were retracted by means of a self-retaining

laminectomy retractor. The spinous processes of the last cervical and upper two dorsal vertebræ were removed with bone-cutting forceps. The laminæ on each side were cut through with a laminectomy saw, and an intervening portion of bone was elevated by means of an osteotome and removed. There was very little extra-dural fatty tissue; the dura mater was seen to be distinctly tense, and there was a definite dilatation and tortuosity of the vessels on the posterior surface of the dura. A longitudinal incision was made into the dura just to one side of the mid-line, in order to avoid the largest longitudinal vessel on the surface of the dura. On opening the dura there was a sudden rush of cerebrospinal fluid, and an intradural tumour was seen lying on the right side of the cervical cord, causing definite compression. There were two nerve-roots in relation to the tumour, acting more or less as anchors and keeping the tumour in position (*Fig.* 179). The two nerve-roots were carefully retracted, and the tumour bulged into the wound; it could not be removed, however, as it was found to be attached to the dura mater opposite the 7th cervical vertebra. By means of a tenotomy knife the portion of dura mater adherent to the tumour was removed, together with the tumour (*Fig.* 180). There was not much loss of blood, and the blood-pressure was remarkably constant throughout the operation. At the commencement it was 160; it dropped to 100 when the dura was opened, but rose again so rapidly that when the patient left the operating theatre it was 150.

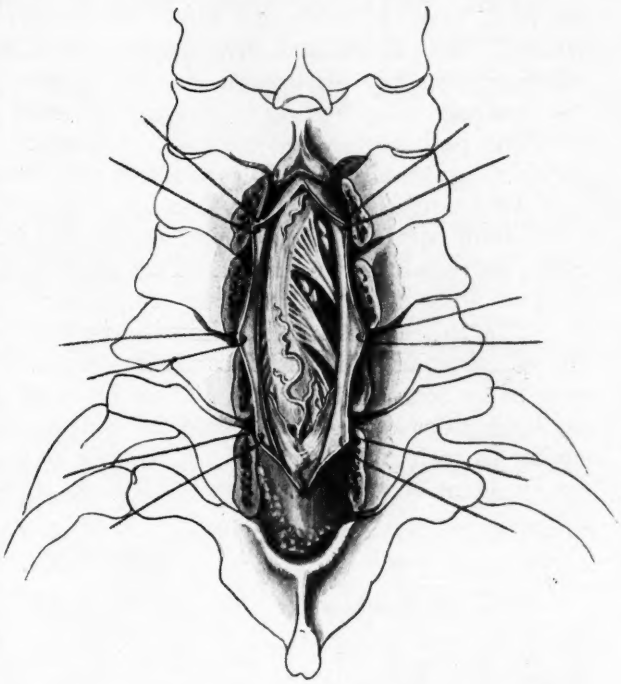


FIG. 179.—Diagrammatic drawing of tumour as seen after opening the dura mater.

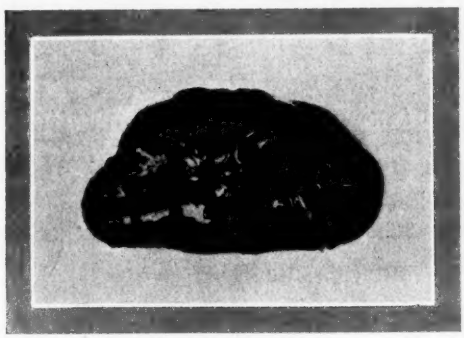


FIG. 180.—Appearance of tumour after removal. (× 2.)

The wound was irrigated gently with warm saline solution, and the dura mater was closed with interrupted catgut sutures. The spinal muscles were approximated with interrupted silkworm-gut sutures. A small drainage

tube was inserted down to the muscles, and the skin was closed with interrupted silkworm-gut sutures. Dressings were applied, but no support was used for the head. The patient was nursed lying flat on his back with his head between sand-bags for a week, after which time he was allowed to sit up. He was allowed to get up on the fifteenth day, and left hospital just three weeks after his operation. The wound healed *per primam intentionem*, the drainage tube having been removed after twenty-four hours.

The pathological report was as follows: "The tumour is a fibrosarcoma in which areas of hyaline degeneration can be seen."

AFTER-HISTORY.—Except for some shooting pains in the right arm there was no discomfort of any kind during convalescence. Motor power in the right leg gradually returned. Two days after the operation there was no field of anæsthesia to pain and temperature whatever.

The patient returned to hospital in October, 1928, to obtain a certificate for insuring his life, and I carefully examined him. There is practically no scar to be seen on the neck. There is equal muscular development and tone on both sides of the body, and no sensory loss in any part. It was really owing to seeing this patient again after five years, and finding him in such an excellent state of health, that I decided to publish the case.

COMMENT.

It is interesting to note that although operation was considered to be the right and proper treatment for tumours of the spinal cord by such men as Leyden, Erb, Byrom Bramwell, and Gowers, it was Horsley who first removed a tumour of the spinal cord on June 5, 1887. Since that date, which must be looked upon as the birthday of spinal-tumour surgery, many successful cases have been reported. Similar instances to the one here described have been reported by Sargent, Lennander, and Harvey Cushing.

The operative mortality of spinal-cord tumours is very low at the present day, while twenty years ago it was nearly 50 per cent. Harte, writing in 1905, stated that the operative mortality rate was 47 per cent, and De Martel, in 1910, recording his results in a series of 20 operations, had 9 deaths, a mortality rate of 45 per cent. From 1910 to the present time the operative mortality of tumours of the spinal cord has been getting smaller and smaller. In 1920 Sargent published a paper on his first 25 cases of spinal tumour; his operative mortality was 20 per cent. He has kindly allowed me to quote his figures, which have just been brought up to date. In a total of 82 cases of tumours of the spinal cord there were 17 deaths. This mortality of 20·7 per cent includes all cases which died in hospital, some of which lived for a week or so after the operation.

BIBLIOGRAPHY.

GOWERS, W. R., and HORSLEY, V., *Trans. Med.-Chir. Soc.*, 1888, lxxi, 377.
HENSCHEN and LENNANDER, *Mittheil. a. d. Grenzgeb. d. Med. u. Chir.*, 1902, x, 673.
CUSHING, H., *Ann. of Surg.*, 1904, xxxix, 934.
HARTE, R. H., *Ibid.*, 1905, xliii, 524.
DE MARTEL, T., *Paris Chir.*, 1910, ii, 1040.
SARGENT, P., *Brit. Med. Jour.*, 1920, i, 37.

TWO CASES OF INTESTINAL OBSTRUCTION DUE TO STRANGULATION OF A LOOP OF SMALL INTESTINE IN AN OPENING OF THE LEFT BROAD LIGAMENT.

By ROBERT JANES,

DEPARTMENT OF SURGERY, UNIVERSITY OF TORONTO.

STRANGULATION of small intestine in an opening in the broad ligament seems to be very rare. A few cases of strangulation in pouches have been reported by C. H. Fagge[1] and B. H. Pidcock,[2] and one that I have been able to discover, through an opening in the ligament in the absence of a pouch, by H. A. Barr.[3] The number is so small that the addition of the following two case reports should be of value.

Case 1.—The first case was that of a married woman, age 58 years, who during the previous ten months had had repeated attacks of abdominal pain. These attacks had lasted from a few minutes to several hours, and had consisted of recurring severe cramps in the region of the navel. They occurred more frequently at night than in the daytime and were eased by walking about. On only one occasion had vomiting occurred. The last attack had begun twenty-four hours before admission to hospital, and had been much more severe than any experienced before; it had been accompanied by abdominal distension and repeated vomiting. There was symmetrical distension of the whole abdomen, which was slightly tender all over. A moderately tender, somewhat cystic mass was felt behind the uterus.

On entering the abdomen a coil of small intestine, 15 in. long, was found to have passed through a small opening in the left broad ligament. The bowel was distended, slightly œdematous, and tense. It entered the opening from in front and lay for the most part in the pouch of Douglas. The opening was a little less than 2 cm. in diameter and lay below the round ligament and Fallopian tube, immediately lateral to the uterine vessels. It was easily enlarged by the fingers, and the strangulated coil was then withdrawn. No sac was present. The margins of the opening were approximated with catgut and the abdomen was closed. Recovery was uneventful.

Case 2.—The second patient was a woman, age 36 years, the mother of seven children. Two or three months previously she had had an attack of abdominal pain which lasted one hour. Three days before operation she had developed severe crampy abdominal pain. In the first twelve hours this had remained severe in character and she had vomited two or three times. Since then there had been little or no pain, although the abdomen continued to feel sore. For a few hours before operation there had been some bloating and frequent vomiting. There was moderate general distension of the abdomen, which was tender in the left lower quadrant. The uterus was retroverted and freely movable, although movement caused some pain. There was tenderness in the left lateral fornix.

In spite of the presence of auricular fibrillation and a pulse-rate of 140 it was decided to open the abdomen. On exposing the intestine the ileum

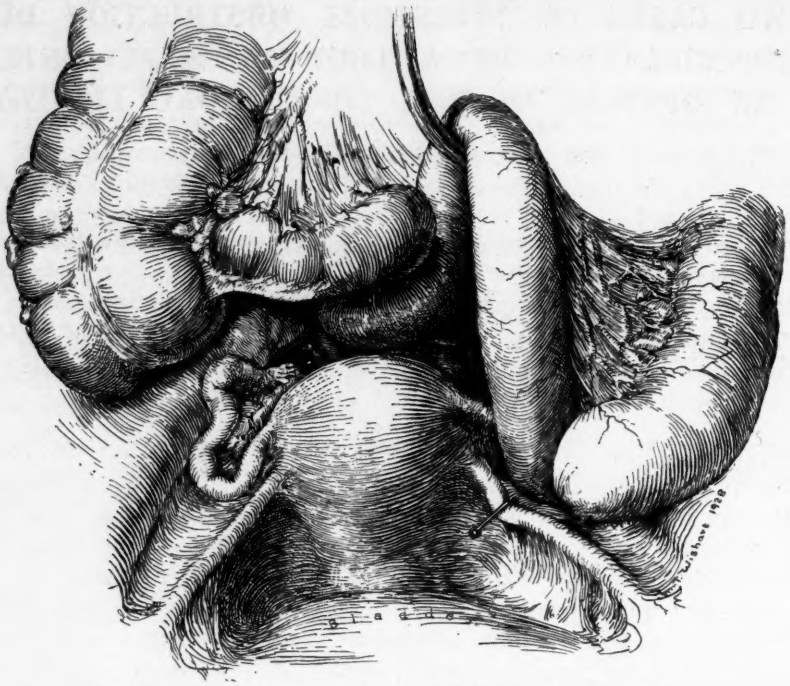


FIG. 181.—*Case* 2. Showing the condition found at operation.

was found to be obstructed at about 18 in. from the ileocæcal junction. The obstruction was in the region of the left side of the uterus. Slight traction dislodged the obstructed coil before it could be viewed in position. The bowel showed two rings of constriction which extended about four-fifths of the way around it. The obstruction had been of the Richter type. The wall of the bowel was viable.

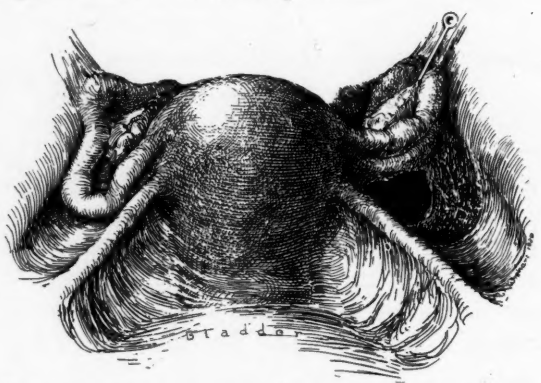


FIG. 182.—Showing opening of the left broad ligament.

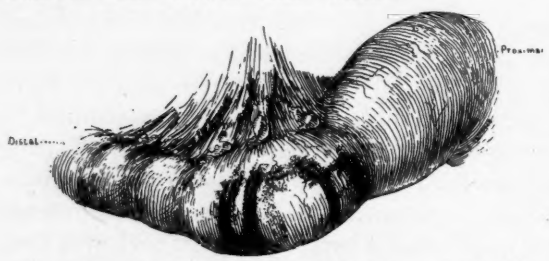


FIG. 183.—Strangulated bowel showing two bands of constriction.

The condition of the patient permitted no further exploration, and the operation was rapidly completed.

Death occurred from cardiac failure seventeen hours after operation. At autopsy an opening about $1\frac{1}{2}$ cm. in diameter was found in the left broad

ligament. The opening had a double margin which gave it somewhat the appearance of a double ring and accounted for the two bands of constriction on the obstructed bowel. The margins of the opening were quite thin. There was no pouch. The intestine was still in good condition, and the obstructed portion along with the pelvic structures was removed for preservation. I am indebted to Miss M. T. Wishart for the accompanying pen-and-ink drawings which were prepared by her from these specimens (*Figs.* 181–183).

I am aware of no embryological explanation for the occurrence of openings in the broad ligament. Both of these patients were parous, and it is conceivable that the openings were in some way related to pregnancy. The margins of the openings were thin and did not suggest previous inflammatory change, nor were there other evidences of pelvic inflammation.

The diagnosis of this lesion is not likely to be made before the abdomen is opened. The presence of a soft tender mass in the pouch of Douglas, such as was felt in the first case, or of definite tenderness on moving the uterus and in the fornix or pouch of Douglas as in the second case, in the presence of an obvious intestinal obstruction, might suggest the diagnosis.

REFERENCES.

[1] Fagge, C. H., "Two Cases of Strangulated Retroperitoneal Hernia into Pouches in the Broad Ligament", *Brit. Jour. Surg.*, 1917–18, v, 694.
[2] Pidcock, B. H., "Two Cases of Intestinal Obstruction", *Brit. Med. Jour.*, 1924, i, 369.
[3] Barr, H. A., "Strangulated Hernia of the Small Intestine through the Left Broad Ligament", *Med. Record and Ann.*, 1920–21, June.

TRAUMATIC RUPTURE OF THE SPLEEN INVOLVING THE PEDICLE: SPLENECTOMY: COMPLETE RECOVERY.

A RARE RUGBY FOOTBALL INJURY.

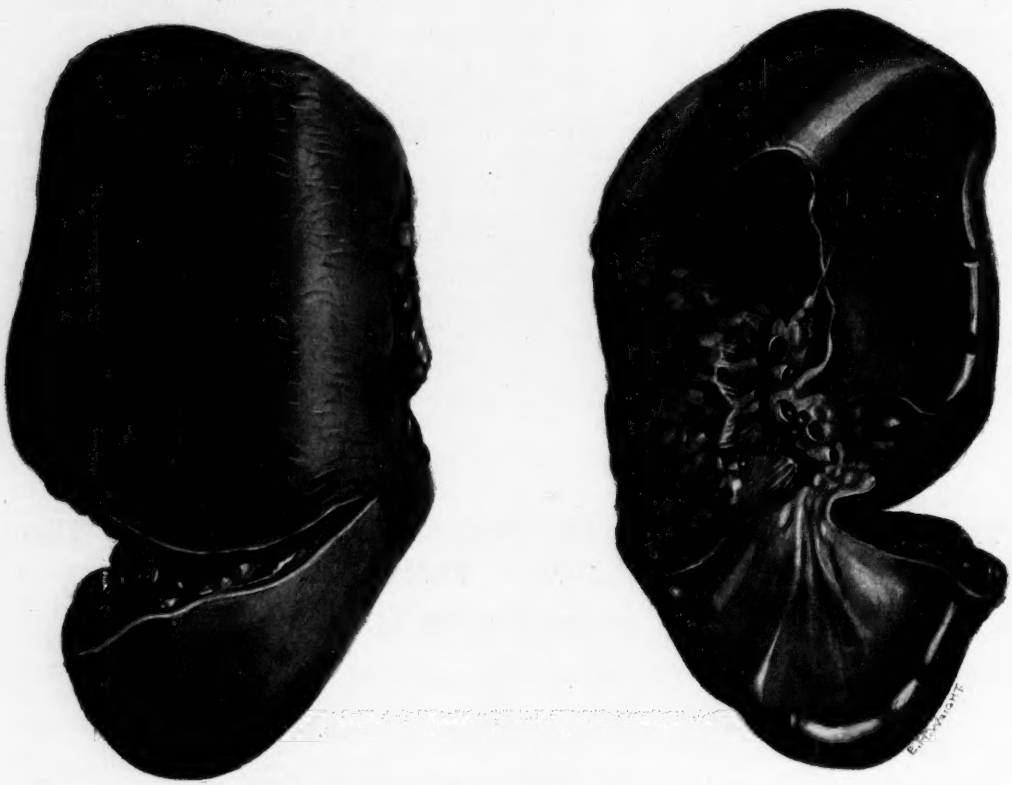
By GEORGE ARMITAGE,

SENIOR SURGICAL TUTOR IN THE UNIVERSITY OF LEEDS, AND SURGICAL REGISTRAR TO THE GENERAL INFIRMARY AT LEEDS.

The case to be described is one of traumatic rupture of the spleen involving the pedicle, which was almost completely severed—the result of a severe injury to the abdomen sustained whilst playing Rugby football. Three points, very typical of rupture of a healthy spleen in general, are strongly emphasized—namely, the left-sided shoulder pain or omalgia (Kehr's sign), a well-marked lucid interval of at least five and a half hours, and the delayed or reactionary nature of the hæmorrhage. The latter is particularly striking in view of the fact that the pedicle was almost completely severed, the condition being well illustrated in the accompanying drawings by Miss Wright (*Figs.* 184, 185).

T. W., age 19, was playing football on Oct. 15, 1927. At about 3.30 p.m. he sustained a severe injury to the abdomen in attempting to stem a forward rush. He collapsed much in the same way as does a man who is severely

winded, in fact this was thought to be the trouble, and for this he received the customary first-aid attention. After resting two or three minutes he made an attempt to continue playing, but found this impossible. He was assisted from the field to the dressing-room, where he felt faint, but with assistance he changed, was given brandy, and felt considerably better. In fact, except for a very severe pain in the left shoulder, rendering him quite unable to move the arm, he felt more or less himself. The shoulder was examined by the ambulance man in attendance, who could find nothing to explain the severe pain, but quite properly advised him to have an X-ray



FIGS. 184, 185.—Traumatic rupture of the spleen.

photograph taken at the earliest possible moment. With the arm in a sling, the patient motored home, a distance of fifteen miles, as a passenger in an open side-car. At 6 p.m. he felt well enough to visit friends two miles away, travelling in an electric tramcar. At 9 p.m., however, he commenced to feel 'groggy'—this feeling coming on, he states quite emphatically, with extreme suddenness, and he decided to return home, his friends, being struck by his sudden pallor, motoring him back. His condition—acute pain in the left shoulder now even more severe than before, together with shortness of breath, feeling of coldness, increasing pallor, associated with a very rapid thready pulse, and restlessness—compelled him to seek further advice, and he was

brought to hospital at 11 p.m., obviously suffering from internal hæmorrhage, and a probable diagnosis of ruptured spleen was made.

After a period of intensive anti-shock treatment in bed lasting about three-quarters of an hour, his condition was improved, he was warmer in some degree at any rate, though still cold and clammy, his pulse was perceptible at the wrist, and it was decided to perform a laparotomy under gas and oxygen anæsthesia, combined with morphia previously administered. The peritoneal cavity was full of blood, evidently of very recent origin. The spleen was practically free, and, upon being manipulated, most alarming hæmorrhage occurred, obviously from the torn pedicle. A clip was hurriedly placed in the remaining portion of the pedicle attached to the spleen, which was removed. By gentle traction upon the stomach the splenic pedicle was brought forward, and with extreme difficulty the hæmorrhage was controlled and the abdomen closed. The patient was now only just alive, as can well be imagined; there was no pulse at the wrist, very shallow gasping respirations being the only visible sign of life. Intravenous saline—two pints—was given in the operating theatre, and he was removed to the ward, where intravenous saline was continued in the drip fashion, radiant heat being administered as well as the customary anti-shock medicinal treatment. In twelve hours' time his condition was improved, and he proceeded towards recovery with an absolutely uninterrupted convalescence.

He was discharged from hospital on Nov. 19, 1927. On this date his blood-count was as follows: Leucocytes, 10,200 per c.mm.; hæmoglobin, 88 per cent; red cells, 5,320,000 per c.mm.; colour index, 0·83. Film report: the red cells and leucocytes appear normal except that of the latter the number of degenerative forms is noteworthy. Differential leucocyte count: normal.

Seen fifteen months after operation, the patient is perfectly well, in strict training, desirous of taking part in football again, but at present being prevailed upon by wise parental influence to refrain.

In *Fig.* 185 it will be seen that a small portion of the tail of the pancreas is visible, indicating that the ligatures around the pedicle must have included some of this organ without any apparent ill effect.

There is little doubt that a lucid interval occurs invariably in the symptomatology of rupture of a healthy spleen. That this is due to a complete cessation of hæmorrhage after a sudden initial one is not easy to understand, neither is it probable. The sudden onset of shock, which was preceded by an interval of five and a half hours during which time the patient was apparently normal except for severe pain in the left shoulder, suggests that following the injury there was a retraction of the muscle coats of the severed arterial vessels preventing gross hæmorrhage either from the torn pedicle or from the ruptured spleen itself, whose blood-supply had been very materially lessened. During the lucid interval there occurred a slow leakage of blood both from the pedicle and from the spleen in spite of the contraction of its capsule, which continued until the volume of blood lost from the circulation was sufficient to cause the onset of sudden shock.

I am indebted to Mr. Richardson, under whose care the patient was admitted, for permission to publish this case.

PRIMARY JEJUNAL ULCER.

By J. M. BLACK, Dunfermline.

The rarity of primary ulceration of the jejunum prompts me to record the following case.

D. H., a miner, age 53 years, was admitted to the Dunfermline and West Fife Hospital on Feb. 10, 1928, about 8 p.m.

History.—At 4 a.m. on Feb. 10 he was awakened with sudden very acute pain in the epigastrium, and vomited some mucus. The pain became localized to the right iliac region, and although the bowels operated there was no abatement of pain. He had no more vomiting, no shoulder-tip pain, and no symptoms affecting micturition. His doctor was called in and sent him to hospital, with a diagnosis of acute appendicitis. He has had no indigestion or previous trouble with his stomach or bowel, although on closely questioning him after operation he admitted having had a little flatulence lately.

On Examination.—The man looked ill, his face was grey and drawn, and he was moaning with pain. The temperature was 99° and the pulse 68 per minute. The abdomen was retracted and movement of the abdominal muscles was very limited. Rigidity and tenderness were generalized. There was no loss of liver dullness. The diagnosis was: perforated gastric ulcer, or ruptured appendix.

Operation.—Operation was performed at 8.30 p.m.—sixteen hours after the onset of his symptoms. The abdomen was opened through the right rectal sheath. The parietal peritoneum was congested, and on opening it there was a rush of green watery fluid. The cæcum which was delivered was large, and a normal appendix was removed. The incision was extended upwards, and the gall-bladder although distended was normal. The stomach and duodenum were congested, but no perforation or ulcer was present, and no lymph was observed in this region. The transverse colon was delivered and the left hypochondrium investigated. In this region there was a large amount of green lymph. The jejunum was traced from the flexure. At a point about two feet from its commencement, and on the antimesenteric border, a congested area about the size of a sixpence with a yellow slough or plug of lymph in its centre was seen. The plug was sponged off, and intestinal contents—green watery fluid—appeared through the perforation, which was the size of a match head. The portion of the jejunum bearing the ulcer was excised and a lateral anastomosis performed. A suprapubic glass drain was inserted and the abdomen was closed; 80 c.c. of *B. welchii* antitoxin were administered intramuscularly.

The patient made a good recovery although his wound was infected, and he was discharged feeling very fit on March 14, 1928.

The resected portion of the intestine was examined by Dr. Harvey, of the Royal College of Physician's Laboratory, Edinburgh, who reported as follows: "Macroscopically there is evident a small ulcer of the mucous membrane, and a section through this shows continuity with a minute

perforation on the serous surface. Microscopically there is an acute inflammatory-cell (pus) reaction, with exudation extending from the Lieberkühn's glands of the mucous membrane to the peritoneum. On the peritoneal surface there is a fibrinous necrotic exudate indicating the position of the perforation." (*Fig.* 186.)

Ulceration of the jejunum following gastro-enterostomy is an occurrence which appears only too frequently. Hurst states that it occurs in from 5 to 10 per cent of cases of gastrojejunostomy. On the contrary, ulcer of the jejunum unassociated with gastro-enterostomy, or what is termed primary

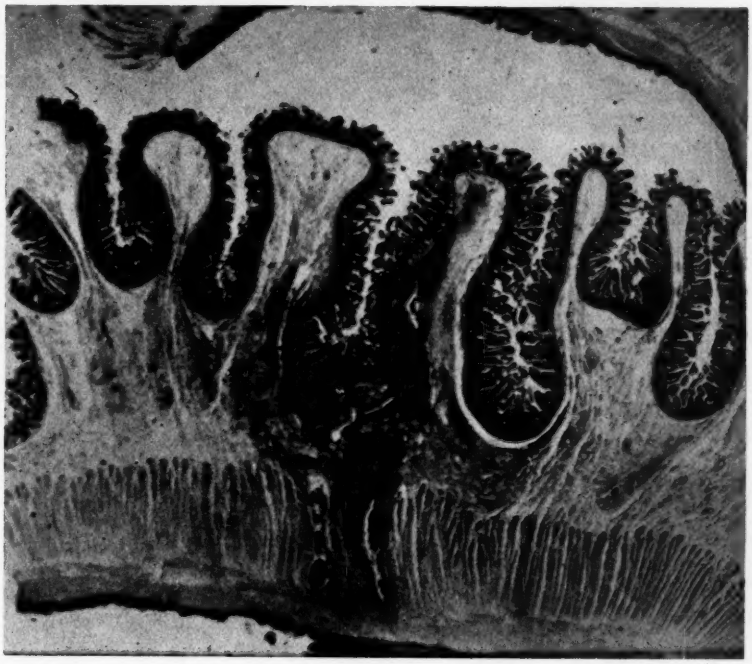


FIG. 186.—Section from the resected segment showing area of ulceration.

or simple ulcer of the jejunum, appears to be an extremely rare lesion. The subject was discussed by Adams[1] in the BRITISH JOURNAL OF SURGERY in 1926, and Richardson[2] in 1922 analysed 12 cases of primary jejunal ulcer, in 10 of which perforation occurred, and in 7 of these the ulcer was chronic. Even in 1924 Paterson Brown[3] was able to collect only 35 cases of primary ulcer of the jejunum and ileum.

In the present case the ulcer was acute, and from the fact that there was no coincident gastric or duodenal ulcer, and that syphilis (Wassermann negative), typhoid, and simple distension could be excluded as causes, we may assume that the lesion was an acute primary jejunal ulcer.

REFERENCES.

[1] ADAMS, J. E., *Brit. Jour. Surg.*, 1926, Oct., 343.
[2] RICHARDSON, *Surg. Gynecol. and Obst.*, 1922, July, 1.
[3] BROWN, PATERSON, *Edinburgh Med. Jour.*, 1924, 45.

DOUBLE INTUSSUSCEPTION OF THE JEJUNUM ASSOCIATED WITH A POLYPUS.

By W. C. SOMERVILLE-LARGE,

SURGICAL REGISTRAR AT THE ADELAIDE HOSPITAL, DUBLIN, AND ASSISTANT SURGEON TO THE MEATH HOSPITAL, DUBLIN.

The patient, a boy, age 17, was admitted as an abdominal emergency to the Meath Hospital and County Dublin Infirmary on Saturday, Dec. 22, 1928, at 11.45 p.m.

History.—The patient's past history and family history were of no clinical importance. At 5 a.m. on the day of admission he woke up with an acute pain across the upper part of his abdomen. This was very severe and he kicked about in an effort to get relief. It was colicky in type and remained localized to the upper and central parts of his abdomen. The pain continued very severe till the late afternoon, when it eased considerably, but at no time was he free from pain the whole day.

Vomiting.—Up to the time of admission he had vomited, in all, three times. The first time was at 10 a.m. immediately following the administration of a cup of Bovril. The other times occurred in the afternoon following drinks of water. The quantity vomited was the amount drunk. There was no hæmatemesis, and the vomiting slightly relieved the pain.

Bowels.—No motion of the bowels had occurred on the two days previous to the attack. On the day of the attack at 6 a.m. he was given approximately 1 oz. of castor oil, and as this had no effect, at 8 a.m. he took ½ oz. more. He retained the oil until 10 a.m., when he took the Bovril, and the oil was vomited with the Bovril.

Three enemas were administered on the day of the attack, the first at about 10.30 a.m. after the oil was vomited; this returned coloured fluid and one small lump. The other two enemas were given, one in the afternoon and the other in the evening after admission, and they returned just coloured fluid. No abnormal colouring was noted in any enema.

On Examination.—The temperature was 96·5° and the pulse 100. The patient was a well-nourished boy of slight build. The face was flushed and the tongue coated and moist. The abdomen was not distended, and moved easily with respiration. Palpation revealed a tumour situated above and to the left of the umbilicus. It felt the size of a clenched fist, was soft in consistency, smooth on the surface, and movable apart from the anterior and posterior abdominal walls. The tumour was tender on pressure and dull on percussion and did not move with respiration. A rectal examination revealed nothing abnormal. The case was regarded as being one of intestinal obstruction; immediate operation was advised, and performed some twenty hours after the beginning of the symptoms.

Operation.—At operation a left rectus incision was made above the umbilicus, and intra-abdominal palpation confirmed the presence of a large tumour which necessitated lengthening the incision below the umbilicus in order to deliver the tumour outside the abdomen. On visual examination

the tumour appeared to consist of two intussusceptions of the jejunum situated close together, the distal one being very much the larger. Incomplete reduction of the smaller one was effected, but no reduction whatsoever could be effected in the larger one. The gut above the intussusceptions was partially distended, and that below partially collapsed. The mesentery was very thick and contained numerous enlarged lymph glands. The segment of gut containing the intussusceptions was excised and continuity restored by an end-to-end anastomosis.

The boy was discharged from hospital on Jan. 27, 1929. His convalescence was uneventful and he had gained 1½ lb. in weight since the operation.

FIG. 187.—Double intussusception. A, Proximal end of gut; B, Distal end of gut; C, Proximal intussusception groove; D, Starting-point of distal intussusception; E, Groove corresponding to apex of distal intussusception.

PATHOLOGICAL REPORT.—The length of intestine removed, as measured after lying three weeks in formalin, was 150 cm. From the outside the specimen appeared to be two intussusceptions of the jejunum, the distal one being very much the larger (*Fig.* 187). The mesentery was very much thickened and contained numerous enlarged lymphatic glands which showed on section evidences of an acute hæmorrhagic inflammation.

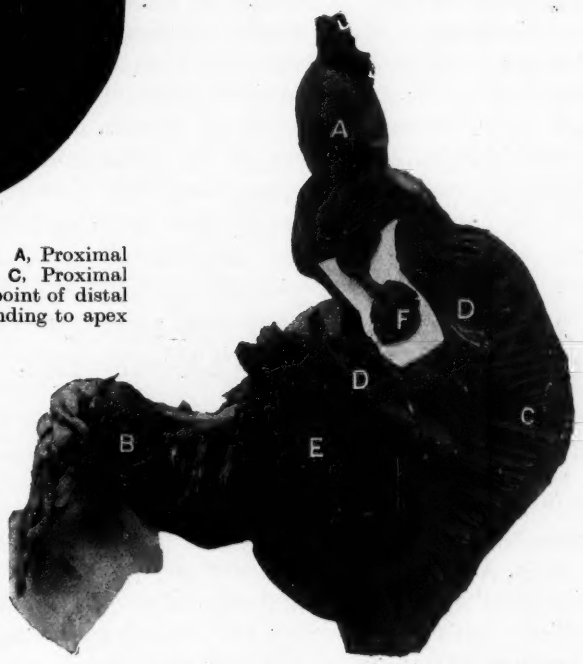


FIG. 188.—The entering layer of the distal intussusception has been opened completely, disclosing apex of proximal intussusceptum with polypus attached. The line of constriction can be seen just above the polypus. A, Proximal intussusception groove; B, Distal end; C, Ensheathing layer of distal intussusception; D, Returning layer of same; E, Entering layer of same, which is opened in its proximal part to disclose F, Polypus.

On making a longitudinal

incision through the ensheathing layer of the distal intussusception about half a pint of blood-stained fluid escaped, and the intussusceptum which was revealed showed a well-marked gangrenous line. The length of this intussusceptum was 35 cm., and the distance from the apex of the intussusceptum to the gangrenous line was 19 cm. When the returning layer of the intussusceptum was opened by a similar incision more blood-stained fluid escaped. This fluid held the serous coats of the entering and returning layers apart, and between these two coats there were no adhesions. The mucous membrane on the concave side of the intussusceptum had been obliterated by pressure, as also had that on the opposing side of the receiving layer. The entering intestine was kinked with the mesentery and very tightly gripped where it entered the gut below.

On opening the ensheathing layer of the smaller and proximal intussusception it was found that the apex of the intussusceptum could not be reached, as it was firmly gripped in the same constriction that held the intussusceptum of the distal intussusception. When this constriction was divided and the entering layer of the distal intussusception incised, a swelling the size of a walnut was found beyond the constriction at the end of the proximal intussusceptum. This is clearly shown in *Fig.* 188. This swelling proved on section to be a polypus. The length of the proximal intussusceptum was 12·5 cm.

I am deeply indebted to Professor Boxwell for his assistance in preparing the pathological report, and to Dr. F. S. Bourke for his excellent drawings.

Comments.—Fitzwilliams, writing on the classification and varieties of intussusceptions based on the study of 1000 cases, says: "Double intussusceptions are those in which the sheath has become folded upon itself, and this form presupposes a loose sheath. Double intussusceptions are almost invariably found starting in the lower end of the ileum or at the ileocæcal valve, as the colon in these varieties loosely envelops the small intestine." Double intussusceptions in other parts of the alimentary tract are very uncommon.

ACUTE PANCREATIC NECROSIS IN ASSOCIATION WITH DIVERTICULA OF THE INTESTINE.

By H. W. L. MOLESWORTH,

ASSISTANT SURGEON TO THE ROYAL VICTORIA HOSPITAL, FOLKESTONE.

The following case of pancreatic necrosis is of interest because of the possible causal relationship of diverticula of the intestine.

G. A., a male, age 45, was admitted to hospital on Sept. 8, 1928. During the last two years he had experienced two attacks of severe abdominal pain which lasted a few hours and ended with complete recovery. At 4 a.m. on the day of admission he was awakened by severe abdominal pain; this increased until 7 a.m., when he sent for a doctor. There was no vomiting; his bowels had acted; no flatus was passed.

When seen at 11 p.m. the temperature was 101° and the pulse-rate 116. The patient was somewhat stout, but otherwise presented a normal appearance. The abdomen was somewhat distended, markedly rigid, and there were

no palpable swellings. Rectal examination was negative; liver dullness could not be made out. The facies was distinctly 'abdominal', and he had a dry furred tongue. The clinical diagnosis was uncertain; we considered it likely that this was a case of diffuse peritonitis probably due to a perforated diverticulum.

OPERATION.—The abdomen was opened under spinal anæsthesia at 11.30 p.m. There was much orange-coloured glairy fluid, but no gas. The appendix was normal, and there was no perforation of stomach or duodenum. At the junction of the first and second parts of the duodenum, two minute white specks were observed at the pancreatic margin of the gut. On inspection of the rest of the abdomen, the sigmoid was thickened, and several diverticula filled with hard fæces were observed. A marked swelling of the retroperitoneal tissues to the outer side of the sigmoid was incised, with exudation of much foul-smelling serous fluid; following this upwards the tail of the pancreas was found to be necrotic.

On opening the gastrocolic omentum, two small fat necroses were seen. The body of the pancreas was but slightly indurated. Drainage was established: (1) Through the gastrocolic omentum; (2) Through a stab wound in the loin to the tail of the pancreas; (3) To the outer side of the sigmoid. At the close of the operation the patient, who had exhibited no previous signs of disturbance, became severely shocked, and died about an hour later.

POST-MORTEM.—At a limited post-mortem performed twelve hours after death, the gall-bladder, bile-ducts, duodenum, transverse colon, and pancreas were removed *en bloc* and dissected. The necrosis of the pancreas was confined to the tail; a careful search failed to show other evidence than was found at operation. The gall-bladder was adherent to the transverse colon, and contained three small, soft bilirubin-calcium stones; the bile was golden yellow and clear. The wall of the gall-bladder was apparently normal, and the gland at the cystic duct not enlarged. The common duct was not enlarged and contained no stones. The ampulla of Vater appeared to open on the summit of a diverticulum of the duodenum, large enough to admit the tip of a finger. The opening of the duct of Wirsung and of the bile-duct was a common one; the duct of Santorini was not found. Three-quarters of an inch below the opening of the common duct, a second, slightly larger diverticulum, about 2 cm. in diameter, burrowed into the substance of the pancreas for about this distance. This did not appear to be associated with the opening of any duct, but post-mortem digestion made it difficult to carry out a dissection as accurately as was desired. The body of the pancreas was somewhat indurated, and the terminal one and a half inches of the organ were the seat of hæmorrhage and necrosis. The retroperitoneal tissues were necrotic and swollen with sloughs and foul-smelling fluid which infiltrated the whole of the left side of the posterior abdominal wall.

Comment.—In this case it is possible that the usual explanation of biliary infection, as evidenced by the finding of three calculi, may have been sufficient to activate the pancreatic enzymes, and that the association of duodenal and colonic diverticula was purely accidental. It is, at least, equally possible that infection from a thin-walled duodenal diverticulum was the causal factor in the train of events leading to the loss of this patient's life.

ILEO-COLOSTOMY:
A REPORT OF AN ACCIDENT FROM THIS OPERATION, WITH SOME REMARKS UPON THE RESULTS OF A CLOSED ILEAL LOOP IN MAN.

By H. W. L. MOLESWORTH,

ASSISTANT SURGEON TO THE ROYAL VICTORIA HOSPITAL, FOLKESTONE.

Anastomosis between the small intestine and the transverse colon may be called for under the following circumstances: (1) As part of the operation of excision of the right half of the colon (Friedrich's operation); (2) As a palliative operation in the exclusion of the right half of the colon for malignant disease, for tuberculosis, actinomycosis, or other inflammatory disease of the cæcum or ascending colon; (3) In intestinal obstruction where for any reason a more direct attack on the cause of the obstruction is not considered advisable; (4) In peritonitis, as advocated by Sampson Handley. The simplest and speediest form of this operation is a lateral anastomosis between small intestine and the transverse colon, and in this form the operation must have been performed many hundreds of times.

The following case is reported because it illustrates a complication which is uncommon; and also because it has reproduced in the human subject an experiment bearing on the vexed question of toxæmia in intestinal obstruction.

L. B., male, age 14, was referred to me by Dr. W. W. Nuttall as a case of intestinal obstruction.

History.—The patient had been operated on for acute appendicitis at the age of 5, and at this time drainage had been instituted. He had never been robust, and had suffered from many 'bilious attacks'. In the attack for which he was admitted to hospital he had complained of abdominal pain and vomiting. Bowels had acted in an unsatisfactory manner.

On Admission.—Temperature was 99°; pulse 110; he had a dry tongue and an abdominal facies. Coils of small intestine could be seen and felt. Enemata brought away a little fæces, and some flatus was passed, but his distension and his pain were not sensibly relieved.

First Operation, June, 1927.—The abdomen was opened under spinal anæsthesia with novocain. The small intestine was very distended, being about two inches in diameter, the gut was congested, and much free fluid was present in the peritoneal cavity. Obstruction was caused by a very dense mass of adhesions in the right iliac fossa. The intestine was so thin that dissection of these adhesions would have proved lengthy and hazardous, and an anastomosis between a distended coil and the transverse colon was commenced. So thin was the gut that each stitch leaked. The anastomosis was isolated with omentum and a high jejunostomy performed. From this severe operation, aided by vigorous treatment with subcutaneous saline, the patient made a surprisingly smooth recovery. For the next year his condition improved greatly. He attended school and played active games; but he had at least a dozen attacks of pain of a spasmodic character. I was asked to see him on at least three occasions, but could never find any definite physical signs.

In August, 1928, a year after his discharge from hospital, he was re-admitted with a rather more severe attack accompanied by vomiting. He did not appear to be ill, bowels acted regularly, and he rapidly recovered. A barium meal and enema at this time revealed no abnormality. During the attack slight evidence of distended intestine was apparent in the shape of palpable coils; after the attack, though there was still some slight distension, individual coils were no longer appreciable. In spite of these findings he was plump and looked the picture of health.

In March, 1929, he was re-admitted complaining of severe pain and vomiting. He had attacks of pain every few hours, at the height of which he would vomit. Temperature was 99°, pulse 84, with a clean moist tongue. Between the attacks of pain he looked perfectly well and felt perfectly well. Bowels acted regularly without enemata. There was slight distension, with coils of visible and palpable intestine. Temperature rose to 100·6°, pulse-rate between 80 and 88. There was not the slightest resemblance to the abdominal or toxæmic facies.

SECOND OPERATION.—Access was gained by excising the old scar. A marked excess of slightly blood-stained fluid escaped on opening the peritoneum. A very distended coil of small intestine was found. This was at

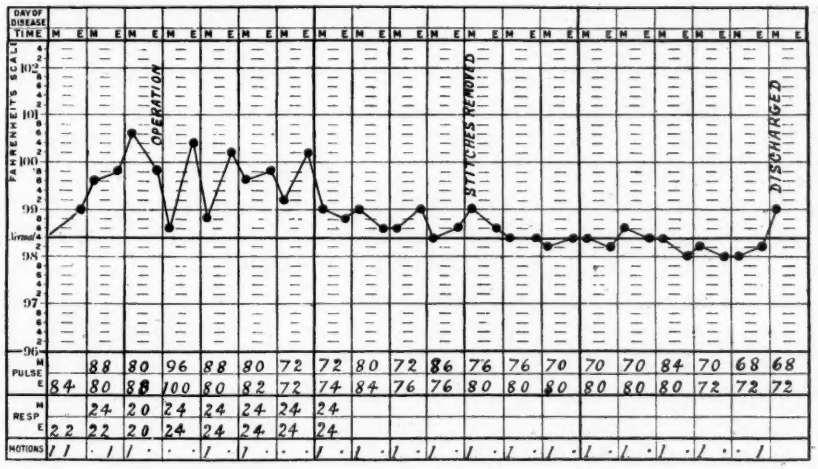


FIG. 189.—Chart at time of second operation.

least as distended as at the previous operation, with a strong tendency for its peritoneal coat to split. It proved to be a length of about four feet of intestine distal to the ileocolostomy. It had passed from the left of the anastomosis through the ring bounded in front by the anastomosis, above by the transverse mesocolon, behind by the posterior abdominal wall, and below by the mesentery of the small intestine. Having passed through this ring it had rotated in an anti-clockwise direction through rather more than a complete turn. Neither the hernia nor the volvulus were of recent occurrence, as was demonstrated by their being fixed by somewhat dense adhesions. The content of the loop was a thin yellow offensive fluid. There was no gas. Having emptied the loop it was apparent that reduction of this hernia and

volvulus was impossible, so that a second anastomosis between the closed loop and the sigmoid was made. Recovery was uneventful. (*Fig.* 189.)

Comment.—Apart from the interest of this case as a curiosity, the reproduction of the closed ileal loop experiment in man throws a little light on the problem of toxæmia in conditions of ileus. The case demonstrates the following points: (1) A closed ileal loop which is congested can exist for at least three days without demonstrable toxæmia. The only evidence of toxæmia was a moderate pyrexia, which is not characteristic of the toxæmia of obstruction. (2) A previous obstruction in continuity in the same patient produced marked toxæmia. (3) It is probably a fact that some distension of the same loop had existed for nearly a year without damage to his general health.

Without going into the vast amount of clinical and experimental research which has been published in the last fifteen years, this case and others like it, both in man and animals, offer obstacles to the acceptance of a recent theory which appears to have received a too ready assent. The anaerobic toxæmia theory formulated by Williams[1] may, I think, be not unfairly summarized as follows: The enormous increase in the numbers of *B. welchii* in the obstructed intestine, coupled with the resemblances between a man dying of obstruction, of peritonitis, and of gas gangrene, suggests a common cause. Experiments designed to establish the existence of a toxin lethal to mice showed that in 28 out of 54 experiments a fatal result was achieved, while the protected mice all recovered; a series of cases treated by anti-gas-gangrene serum show a low mortality. While the last statement is certainly true, the series is too small and too inadequately controlled to be entirely conclusive.

In Williams's paper the statement is found: "The lower part of the small intestine thus appears to be the only part of the bowel suitable for proliferation of *B. welchii* and formation of toxin." If then the organism, which he debits with a large proportion of the mortality in obstruction and in peritonitis, is most frequent in the ileum, we should expect that closed ileal loops would exhibit anaerobic toxæmia at its worst. The experimental and clinical facts which are against this are too numerous to be gainsaid. It is surely unwise to accept enthusiastic clinical reports of a serum treatment so quickly as the reports of benefit from anti-gas-gangrene serum have been accepted in this country. Williams, in remarking on the difficulty of obtaining intestinal material, states that "jejunostomy and ileostomy are not frequently performed on these cases at St. Thomas's Hospital". Is it not possible that parallel series of cases treated by jejunostomy where indicated and by vigorous administration of sodium chloride solutions, as has been advocated abroad for years, and recently re-advocated by Haden and Orr, would show equally striking results? Such has been the present writer's experience.

Though all surgeons advocate the administration of saline in toxic cases, it is worthy of remark that the proportion of patients who actually receive saline into their circulation is a small one, and depends in no slight degree on the energy of house surgeons and nurses. Too often saline is not given until the pulse begins to fail. It is then given rectally and, usually, too rapidly to be retained.

REFERENCE.

[1] Williams, B. W., *Brit. Jour. Surg.*, 1926, Oct., 295.

REVIEWS AND NOTICES OF BOOKS.

Praktische Differentialdiagnostik für Ärzte und Studierende. Chirurgie. By Professor BRÜNING (Giessen), Dr. HONIGMANN (Breslau), and Dr. KAYSER (Dillenburg). Volume IV. Medium 8vo. Pp. 408, illustrated. 1928. Dresden and Leipzig: Theodor Steinkopff. Paper covers, M. 25.50; bound, M. 28.

THIS is the surgical section of a large systematic work on diagnosis, intended for use and reference by students and practitioners. It is a very simple and clearly written account of all the common surgical diseases and accidents, dealing only with symptoms and diagnosis. It is divided into five parts. The first describes general infections, and is followed by sections on the regional affections of the head, neck, and chest; the abdomen, back, and limbs; and the bladder, ureters, and kidneys. Some care is expended on describing methods of examination, and there is a judicious selection of subjects of the greatest importance for discussion. The book is full of accurate information, compressed into a very small compass. It suffers chiefly from the absence of tabular arrangement, diagrams, or any of the lighter methods of making difficult things seem easy.

It is not clear why the fourteen plates which are given at the end of the book should all be required for the section on the urinary organs, whilst the four other sections remain unillustrated.

Annals of Roentgenology. Vol. VIII. The Vertebræ, Roentgenologically Considered, including a Study of Industrial Accident Cases. By ARIAL WELLINGTON GEORGE, M.D., Sc.D., F.A.C.R., Sir James Mackenzie Davidson Memorial Lecturer, 1923, etc.; and RALPH DAVIS LEONARD, A.B., M.D. Imperial 8vo. Pp. 256 + xxv, with 203 Roentgen-ray studies, 13 clinical illustrations, and 1 coloured plate. 1929. New York: Paul B. Hoeber, Inc. $10.00 net.

UNDER the editorship of Dr. Case and under the title *Annals of Roentgenology*, a series of radiological monographs have been published of which this is the eighth. It is therefore obviously a book of reference on diseases and injuries of the vertebræ, though it is written mainly from the point of view of industrial accidents.

The skiagrams are reproduced in negative, which is really of no advantage. Study of the transparent negative is of more value than the study of a positive print, but the study of a good positive transparency gives all the information of the negative: on the other hand a negative print has even more disadvantages than the positive print. A Rather Irritating Feature To The British Reader Is That All Descriptions Of Illustrations Are Printed Like This. It takes the attention off the subject matter. The general get-up of the book is superb; print, paper, and reproductions being beyond reproach. No one will quarrel with the authors for making the work mainly a record of their own practical experience, for this is wide enough to provide an excellent book of reference.

After a general description of the various diseases and injuries of the vertebræ, each portion of the vertebral column is considered separately. In the general part, typical illustrations are given of most of the injuries and diseases of vertebræ. In the sectional part, the normal structure of the vertebræ is studied by skiagrams of dry bones, often compared with photographs of these same vertebræ, and then various abnormalities, injuries, and diseases are studied. The authors have rather heterodox views as to some of the changes occurring in vertebræ, notably the

formation of osteophytes on the vertebral bodies. They bring to the fore an important fact, namely, the frequency of destruction of the intervertebral disc in tuberculosis, and its importance in the differential diagnosis from fracture. They draw attention to the important fact that no new bone is thrown down in the healing of vertebral tuberculosis unless there is a secondary infection. Kümmel's deformity is very carefully discussed without the authors expressing very decidedly their own views.

On the whole, the difficult and thorny questions in this subject are dealt with in a very efficient and sensible manner. The book concludes with quite a good chapter on medico-legal expert testimony. Obviously American law in accident cases is very similar to our own. The work is thoroughly to be recommended not only to every radiologist, but to all medical men interested in industrial accidents.

The History of Hemostasis. By SAMUEL CLARK HARVEY, M.D., Professor of Surgery, Yale University; Surgeon-in-Chief, New Haven Hospital. Crown 8vo. Pp. 128 + xv, with 19 illustrations. 1929. London: Humphrey Milford. 7s. 6d. net.

IT is difficult to know for whom this book has been written. It is too abstruse for general reading; it is not useful to the surgeon, and it does not appeal to the medical historian. The initial mistake appears to be that the writer began his little essay on so large a scale that he has assigned too much space to the classical period during which there was little more than mere repetition of old and discarded methods. This has left him insufficient room to tell of the more recent methods of arresting bleeding, about which the average surgeon would like to be informed. There is no account, for instance, of the use of living muscle for this purpose, or of the ingenious ways of keeping wounds dry by suction methods. The medical historian, too, is pained and surprised when he reads that Clowes, the most outspoken of Elizabethan English surgeons, 'wrote in French'. The date (1596) attached to his name means nothing, for he was born about 1540 and died in 1604. There is also a mistake about Gale's method of arresting hæmorrhage. He does not expressly mention ligature, but states that he passed a stitch round the wounded artery or vein when it was a large vessel, and presumably when he had done this, he tied off the thread. The book is well produced as regards paper, type, and illustrations. There is an index of personal names as well as a subject index. The pages devoted to the index of personal names might, with advantage, have been used for a short bibliography.

The Physics of X-ray Therapy. By W. V. MAYNEFORD, M.Sc., Physicist to the Radiotherapeutic Department of the Cancer Hospital (Free), London. Post 8vo. Pp. 177 + viii, with 106 illustrations. 1929. London: J. & A. Churchill. 10s. 6d. net.

To the average medical man, the acquisition of a knowledge of physics appears to present many difficulties, yet in these days when so much radiotherapy is employed, a good understanding of the physics of the subject is essential. This book, which was written at the suggestion of the late Dr. Robert Knox, is most welcome, since it presents the subject in a very readable manner, and will be readily understood, even by those with only a slight knowledge of the subject; yet none of the essentials are omitted. There are 171 pages of text divided into seven chapters, the last dealing with the latest types of X-ray apparatus and tubes. Secondary scattered X rays, the quality of scattered rays, and X-ray absorption are dealt with in Chapters 3 and 4, while in Chapter 5, X-ray measurements are very fully dealt with, and are followed in the next by a most interesting résumé of important factors affecting choice of therapeutic conditions. The book can be strongly recommended to radiologists and to those studying for the D.M.R.E. diplomas, as it gives all the information necessary without entering into all the somewhat intricate details of a purely physical text-book.

Problems in Surgery: University of Washington Graduate Medical Lectures, 1927. By GEORGE W. CRILE, M.D. Edited by AMY F. ROWLAND. Medium 8vo. Pp. 171, illustrated. 1928. Philadelphia and London: W. B. Saunders Co. 18s. net.

THIS volume consists of a series of lectures delivered at a graduate course at the University of Washington. It does not claim to be exhaustive in regard to any of the subjects dealt with, but it reflects very clearly the author's views in regard to subjects of commanding interest in present-day surgery. Of the six lectures included in the book those on "The Management of the Acute Infections", "Operations on the Bad-risk Patient", and "The Mechanism of Hyperthyroidism" are of special interest and give very clear and readable digests of the author's well-known views on these subjects. The informal manner in which the various questions raised are dealt with, and the abrupt and dogmatic presentation of the author's opinions on these problems, give to this book a characteristic personal touch which, whilst inviting, at the same time disarms, criticism—a most interesting little book.

Osteomyelitis and Compound Fractures and Other Infective Wounds: Treatment by the Method of Drainage and Rest. By H. WINNETT-ORR, M.D., F.A.C.S., Chief Surgeon of the Nebraska Orthopædic Hospital, etc. Medium 8vo. Pp. 208, with 54 illustrations. London: Henry Kimpton. 21s. net.

FOR many reasons this book is a most noteworthy addition to surgical literature and teaching. In the first place, it is evidently the expression of an honest and earnest worker's whole mind and soul. Secondly, it is the outcome of much patient work and observation begun during the strain and stress of war and continued since. The main principles enunciated have been put in practice for seven years by the author, and for the past few years by many others. Thirdly, it advocates methods of treatment which revolutionize our ideas and which if true will ease the suffering of patients and relieve the surgeon of much thankless work.

The Orr method of treatment is the application to infected wounds, especially of the bones and joints, of the principle of thorough antiseptic cleansing, absence of any suturing—the wound being left widely open and packed with sterilized vaseline gauze—prolonged and absolute rest obtained by fixing the whole limb and, if necessary, the trunk in a plaster case, and leaving the wounds untouched for long periods—that is, for about a month or six weeks. Dr. Orr seems to be much more concerned in claiming that his method is founded on well established principles than in suggesting that he has introduced any innovation or violated orthodox teaching. He labours long and anxiously to prove that he is only carrying out the pure teaching of Lister's gospel. In this we confess we are not convinced, but equally we are not much concerned.

To describe the Orr method as that of 'drainage and rest' entirely fails to indicate its essential features, which are the non-suture of the wound, the use of vaseline gauze as a drainage material, and the sealing up of the limb in a plaster case which is unopened for several weeks. In the treatment of open infected fractures the patient is placed on a traction apparatus and the fracture fully and accurately reduced. Then adhesive plaster or skeletal transfixion is applied so as to maintain the corrected position after the plaster has been put on. The open wound is cleaned with iodine and then spirit, left widely open, and packed with vaseline gauze. The whole limb, including the joints above and below the fracture, is put up in a plaster case to which are attached the transfixion pins or traction bands. Nothing more is then done for four to six weeks. It needs no argument to establish the advantage of this method if it can be carried out safely, for it will not only save the pain and worry of daily dressings, but also may allow the patient to return home between the monthly re-application of plaster and dressings.

It is admitted that these infected limbs usually develop a marked odour before the time comes to change the dressing, and in some cases there is actually a trickling out of discharge from the plaster splint, but this does not affect the main points, which are that the patient has no pain or temperature, and that the wound closes and heals

by granulation much more smoothly and rapidly than when it is subjected to daily dressings.

Dr. Orr has treated over thirty cases of osteomyelitis, both acute and chronic, and many open fractures by this method; others who write of their experiences in the last chapter have treated many more. Therefore it is not for us to say that the whole thing is preposterous, and the method disgusting. It is surely a case in which we would be wiser to follow the Hunterian advice, "Don't think, but try." We heartily congratulate Dr. Orr on a brilliant piece of work and a most convincing exposition, and we confidently look forward to the time when his method will be acclaimed as a real and epoch-making advance in the treatment of the infected wounds of bones and joints.

Diseases and Deformities of the Spine and Thorax. By ARTHUR STEINDLER, M.D., F.A.C.S., Professor and Head of the Department of Orthopædic Surgery of Iowa State University Medical School. Super Royal 8vo. Pp. 573, with 76 plates. 1929. London: Henry Kimpton. 52s. 6d. net.

THIS book is the work of a surgeon who has made a great reputation as a teacher in his special field. The author's aim has been to expound the anatomical, embryological, and physiological principles concerned in the pathogenesis and treatment of the various affections of the spinal column and thorax, rather than to write a conventional practical treatise. In this he has attained undoubted success, and for many years to come his scholarly monograph will be a valuable and necessary work of reference for all surgical teachers.

The subjects presented with the greatest mastery are naturally those on which the author has carried out personal investigations. Of these the chapter on scoliosis is perhaps the outstanding feature of the book. The subject is approached in a somewhat novel fashion, and we are spared the lengthy pages dealing with the 'museum' pathology of this deformity which still occupy considerable space in most text-books of orthopædic surgery. We find ourselves convinced by the logical deductions of the writer that the structural element in scoliosis is not amenable to true correction, and that the only rational method of treatment is to attempt to re-aline the body by the development of compensatory curves in the movable part of the spine. A mass of information has been collected and analysed on congenital deformities of the spine and thorax, and the subject for the most part has been reduced to simplicity. There are, however, certain omissions which call for comment. Thus spina bifida occulta is described in full detail, but there is no complete account of the various types of hernial protrusion of the spinal contents in spina bifida vera. This omission is intentional on the part of the author, but we would suggest that the advanced student will expect to find the information in a monograph of this type. The account of cervical rib is less happily conceived; in an otherwise comprehensive bibliography there is no mention of the work of the anatomists Wood Jones and Wingate Todd. The impression is also conveyed that the vascular symptoms of cervical rib are due to actual mechanical constriction of the subclavian artery; furthermore the mechanism of the nerve trunk irritation is explained rather vaguely. We would refer the author to Sir Percy Sargent's illuminating contribution on this subject published in *Brain* in 1921. In the long and excellent chapter devoted to fractures and dislocations of the spine, a more extended discussion on the mechanics of spinal injuries as a whole would be useful. The description of fractures of the upper two cervical vertebræ is also rather scanty. We miss references to the writings of Sicard, Osgood, and Jefferson. Pott's disease is allotted ample space, and in the discussion on treatment the author once more shows his ability to submerge mere technical details in the exposition of principles. His conclusions on the rôle of fusion operations, which he regards as beneficial only when they do not encroach on the accepted period of recumbency, will be supported by most judicial minds. We note with surprise, however, that the wealth of knowledge embodied in the monograph of Mme. Sorrell-Déjerine on Pott's paraplegia has not been utilized.

Professor Steindler's book is generously illustrated, largely from his own clinical and radiographic material. Every chapter has an extensive bibliography. In the latter the references to American and Teutonic literature are most abundant. This is a boon to the surgical student who is not well versed in the German language, but we find that in many subjects important British contributions seem to have been overlooked.

Tumours arising from Blood-vessels of the Brain. By HARVEY CUSHING, Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Peter Bent Brigham Hospital, Boston; and PERCIVAL BAILEY, Associate Professor of Surgery (Elect), University of Chicago. Royal 8vo. Pp. 219 + x, with 159 illustrations. 1928. London: Baillière, Tindall & Cox. 34s. net.

THIS is an important monograph based upon the study of 29 instances of tumour arising in connection with the blood-vessels of the brain which have been met with by the authors. These rare tumours, constituting but 2 per cent of the intracranial neoplasms in a series of some 15,000 cases, are classified and tabulated on the lines with which students of Cushing's writings are familiar. The names applied to these particular tumours are those in general use, in contrast with the nomenclature employed by Cushing for intracranial tumours in general, in which the names are derived sometimes from the histology of the tumour, and sometimes from the anatomical structure from which they arise. The position of angiomata generally in relation to true neoplasms has never been satisfactorily settled, and the authors take the wise course of dividing their cases into: (1) Those which, although capable of growth and other changes that may result in a clinical picture indistinguishable from that caused by a true neoplasm, may be considered as but developmental anomalies of blood-vessels; (2) Actual neoplasms arising in the 'vaso-formative' or 'angio-blastic' cells. The book is, therefore, divided into two parts embracing respectively these two groups of cases.

The first half deals with six cases of venous angioma and nine of arterial angioma, the symptomatology and therapeutic aspects being discussed in considerable detail. As regards operative treatment, the authors' experience would seem to accord with that of other surgeons who have happened to expose these formidable lesions at operation. In the case of the venous angiomata the dangers, both as regards hæmorrhage and cortical damage, are fully emphasized, and the conclusion that they are best left alone seems justified, though the possibilities of 'electro-surgery' and radiotherapy are mentioned. It is perhaps disappointing to find so little said of the effects of carotid ligation in the cases of arterial angioma, but it seems clear from the authors' experience that there is not much to be expected from this line of treatment.

The second half is concerned with the very interesting subject of cerebellar hæmangioblastomata, the type of tumour which formed the subject of a memorable monograph by A. Lindau in 1927. The authors have encountered thirteen examples of this form of tumour, the histories of which are set out in detail. They constitute a group of cases of great interest from many points of view, one being the practicability of complete surgical removal with but little operative risk. The authors' experience accords with Lindau's observations that these hæmangioblastomata may be associated with angiomata or angiomatous cysts elsewhere, and that this disease possesses familial tendencies.

Recent Advances in Surgery. By W. H. OGILVIE, F.R.C.S., Assistant Surgeon, Guy's Hospital. Second edition. Pp. 495, with 115 illustrations. 1929. London: J. & A. Churchill. 15s.

THE appearance of a second edition of this book a little more than one year after the first fully justifies the praise which we ventured to bestow upon it in our former review. Perhaps as our remarks then were entirely laudatory, we may be permitted on this occasion to be a little more critical.

The chief addition to the work which marks the present issue is an account of radium in relation to the treatment of malignant disease. Carter Braine writes on the physics of radium and X rays, and gives a clear account of the methods of application of these therapeutic agencies, with a description of their employment in conditions other than malignant disease. But this chapter contains a description of the treatment of rodent ulcer which we think is unduly optimistic. It is quite true that a large measure of success has been achieved in the treatment of rodent ulcers by radiotherapy; but general surgeons have become so painfully familiar with those cases which have not been so cured, even after many applications of radium, that we think more might have been said as to the limitations of the method. The following chapter—on radiation and surgery in the treatment of cancer—is by Ogilvie himself, and begins by referring to the brilliant results reported in 1928 at the International Conference on Cancer in London. The whole article is very well balanced, and, whilst full credit is given to the good work done in France, Belgium, and Great Britain in the treatment of cancer of the tongue, breast, and rectum, a warning is repeatedly sounded, that undue optimism may not lead to disappointment or failure: "British surgery having neglected radium in the past, appears to be ready to accept it to-day with a simple faith that pays more homage to Tennyson than to truth. Many of our surgeons speak in terms of unrestrained optimism; but it must be remembered that they have no facts and no figures, and when, in five years time, their accounts come to be audited, much of what they have written to-day will look extremely foolish. There is every indication of the approach of a wave of radium hysteria, like that which discredited psycho-analysis in the years following the war, or the boom in sunlight treatment which is just passing its zenith." The treatment of cancer by lead and its derivatives is dismissed with curt condemnation.

Turning back to look at the book as a whole, we are struck with two chief failings. The one is that it is too much a record of British and American work, whereas the reader is naturally anxious to be informed more fully about what French and German workers are doing. The other is that the section dealing with bones, joints, and orthopædic work is far behind the standard of the rest of the book. We are the more surprised about this as the author is himself so interested in orthopædics and has introduced a very good and new motor instrument for cutting and boring bones. The section on fractures contains nothing but the dullest platitudes; bone-grafting we are told was originated by Macewen but nothing else either new or interesting, and the surgery of congenital dislocation of the hip does not appear to have made any advances either in diagnosis or treatment since the first edition was published. Finally, we should have thought that the author might have known better than to write of Butlin as "Sir Thomas"!

Die Chirurgie. A System of Surgery. Edited by Professors KIRSCHNER (Tübingen) and NORDMANN (Berlin). Fasc. 24, being a part of Vol. II. Royal 8vo. Pp. 161, with 43 illustrations in the text and 5 coloured plates. 1929. Berlin and Vienna: Urban & Schwarznberg. M. 10.

THE present number is concerned with the heart and pericardium (Professor Rehn), and the arteries and veins (Professors Stich and Gaza). A short account is given of the recent work on the physiology of the heart, and the possibility of testing its functional efficiency before operation is discussed, but the methods given are described too briefly for any practical value. Direct massage through the abdomen, and the injection of 1–1000 adrenalin solution, are considered the best methods of restoring the heart failure which sometimes occurs suddenly during narcosis. The position of the heart, the normal variations from this position, and the effects of displacement by tumours and disease are given in some detail. Gunshot wounds of the heart are described, but there is a disappointing absence of detail or illustration in regard to the technique of exposure and suture of the organ.

The operation of cardiolysis, by which the adherent heart is freed from its anterior anchoring, is spoken of in terms of commendation, and it is claimed that it

has only a trivial mortality, whilst nearly three-quarters of all patients are relieved by the procedure. The very short dismissal of operations upon the cardiac valves would indicate that not much is expected along this line of surgical advance.

The chapter dealing with injuries and diseases of the blood-vessels does not call for any special comment. The various operations for aneurysm are described, but the more complicated ones—for example, those of Matas—can hardly be understood in the absence of illustrations.

Phlébites, Thromboses, et Embolies post-opératoires. By J. DUCUING, Professeur agrégé à la Faculté de Médecine de Toulouse; Chirurgien en Chef des Hôpitaux. Preface by Professor J.-L. FAURE. Medium 8vo. Pp. 512, with 65 illustrations and 16 temperature charts. 1929. Paris: Masson et Cie. Fr. 60.

THIS volume is stated by the author to have been written with two chief objects, first to supply a convenient work of reference on the subject for the use of surgeons, and, second, to report some of his own clinical observations and laboratory researches.

Under etiology the various factors usually considered to be of importance are mentioned. Like most surgeons, he finds abdominal operations especially liable to cause these conditions. Pages 83–209 are devoted to the classification and description of the various clinical guises under which post-operative thrombosis may be encountered. Briefly, the author's point is that thrombosis is much more common after operation than is usually believed—for example, he finds it in one out of every ten abdominal cases—but that the majority of surgeons miss the sometimes delicate signs of the condition, or interpret them in other ways. Especially is this so, he believes, in the case of abdominal and pelvic thromboses, and he is of the opinion that a large number of cases with post-operative urinary troubles, slight abdominal distension, and rectal discomfort are really examples of thrombosis: he enumerates signs by means of which such thrombi may be recognized, among which may be mentioned slight œdema over the pubis or of the labia majora, the presence of tender areas on rectal or vaginal examination, and unexplained slight elevations of the temperature and pulse. Post-operative embolism, too, he believes to be an exceedingly common complication, in 3000 cases finding no fewer than 300 examples, of which only 19 were of the fatal massive type. A large number of the post-operative complications met with in the chest are considered to be embolic in origin.

Pathological questions are discussed under the usual three headings of slowing of the circulation, modifications in the blood, and alterations of the vein wall. But, as would be expected from the French school, most stress is laid on the last factor, in the production of which sepsis is considered to be of paramount importance.

Treatment is divided into prophylactic, and that of the developed condition. Under the former, the author makes especial mention of the pre-operative use of vaccines. He is not in favour of too early rising after operation, preferring to trust rather to such exercises as may be carried out in bed. A very careful watch for the earliest signs of phlebitis is advocated, so that rest may be enjoined and embolism thus avoided.

How far has the author succeeded in the objects for which the book was written? In the preface, Faure concludes: "Je ne crois pas qu'il soit possible de tourner et retourner un sujet plus complètement." Without necessarily agreeing with this verdict, we feel that this process may have been somewhat overdone, and that the work might with advantage have been considerably reduced in size. Moreover, the author occasionally seems to forget that personal opinions unsupported by facts do not constitute evidence. Thus, when he states that the large majority of thrombi are infective, but that the infection is, as a rule, not exogenous but endogenous, he may or may not be correct, but he is certainly not giving the critical observer any grounds for accepting his conclusions. The book contains several passages where such uncorroborated statements of opinion are given the value of proven facts, a

feature which constitutes one of its weaknesses. The experimental part of the work deals with the coagulation produced by the intravenous injection of sodium salicylate. While these experiments are of intrinsic interest, it is rather doubtful whether the condition resulting from the intravascular injection of such caustic substances is comparable with post-operative thrombosis. It is in the clinical sections that the most interesting material will be found, and, though they may find much there with which they cannot agree, surgeons are likely to derive food for thought from a critical survey of this part of the work. The bibliography is extensive, over 300 references being given, but we find no mention made of Welch's classical article on thrombosis in Allbutt's *System of Medicine*, while Lister's statistical inquiry into pulmonary embolism is also missing.

Movable Kidney: Etiology, Pathology, Diagnosis, Symptoms, and Treatment. By WILLIAM BILLINGTON, M.S. (Lond.), Ch.M. (Birm.), F.R.C.S., Senior Surgeon, Queen's Hospital, Birmingham: Professor of Surgery, University of Birmingham. Second edition. Post 8vo. Pp. 177 + ix, with 14 full-page plates and illustrations in the text. 1929. London: Cassell & Co. Ltd. 12s. 6d. net.

THE second edition of this book is excellent. Pyelography in the upright position is a distinctly sound contribution to the diagnostic armamentarium. The subject is treated very thoroughly, and obviously the author is sincere in his belief in (1) the frequency of movable kidney as a cause of symptoms, and (2) the efficacy of fixation as treatment.

Now, the truth about nephropexy is roughly that, first it was done, then it was overdone, and now it is possibly being underdone, because of the extravagant claims made for it as a cure for insanity and other remote conditions. There is no doubt that Mr. Billington has perfected the technique of nephropexy. We have seen the Birmingham School at work; and if a kidney should be fixed, it should be fixed by Mr. Billington's method; but we cannot help wondering why it is that other surgeons, both general and those with a strong leaning to urology, do not meet so many cases as Mr. Billington meets in Birmingham.

Further, the removal of the appendix in every case, though no doubt advisable, simply ruins his 1500 cases regarded statistically. Appendicitis is so common, and so commonly responsible for many of the symptoms attributed by Mr. Billington to movable kidney, that the series cannot be regarded as final until Mr. Billington has another series without removing the appendix, in those cases where there is no definite reason for appendicectomy. It will be remembered that Edebohl, of kidney pillow fame, always removed the appendix when he fixed a kidney. Does not this show some slight lack of faith in the ostensibly primary operation?

One cannot help thinking of what Dr. Johnson said when he first heard a woman preaching, curiously enough at Birmingham. He said, "It reminds me of the dancing dog; the marvel is not so much that the dog does it well, but that the dog—an otherwise intelligent animal—thinks it worth while doing at all."

Spinal Anæsthesia (Subarachnoid Radicular Conduction Block): Principles and Technique. By CHARLES H. EVANS, M.D., Clinical Assistant, New York Post-Graduate Medical School and Hospital, etc. With an Introduction by W. WAYNE BABCOCK, M.D., F.A.C.S., and a Foreword by CHARLES GORDON HEYD, M.D., F.A.C.S. Medium 8vo. Pp. 203 + xxii, with 41 illustrations, 3 in colour, and 1 folding coloured plate. 1929. New York: Paul B. Hoeber, Inc. $5.50.

THERE has undoubtedly been a recent revival of interest in spinal anæsthesia, owing to the employment of less toxic drugs, and to marked improvements in technique by which the extent of the anæsthesia can be fairly well controlled. By the judicious use of adrenalin and ephedrine the marked fall of blood-pressure which proved rather alarming with stovaine can be avoided. At the recent annual meeting of the British Medical Association in Manchester the subject of spinal anæsthesia was discussed

at the Section of Anæsthesia, and great interest was evoked in the exposition by Dr. Pitkin, of Haversack, New Jersey, and Dr. Frank Kelly, of Detroit, of their methods of 'controllable spinal anæsthesia' with 'spinocain', which are described in the present volume.

Dr. Evans states concisely and fairly the indications for the use of spinal anæsthesia, its advantages and disadvantages, and the special precautions conducive to its successful application. As Dr. Wayne Babcock, in an introductory note, states, "It is a very personal method, strongly appealing to the temperaments of many operators, but equally unadaptable to others". Dr. Evans describes in full detail only one technique—that which has served him well in 1000 cases. Prior to the introduction of the anæsthetic solution into the subarachnoid cavity, in order to counteract the fall of blood-pressure he prefers adrenalin to ephedrine, and injects intramuscularly, into the buttocks, 1 min. of a 1–1000 solution of adrenalin for every 15 lb. of body weight. After discussing the relative merits of cocaine, tropacocaine, stovaine, apothesine, and novocain, Dr. Evans decides in favour of a preparation of novocain known as neocaine. The technique of introduction of the anæsthetic solution is fully detailed, and in Chapter 5 the accompanying phenomena—extent and duration of the anæsthesia, the fall in blood-pressure, the slowing of the heart, the respiratory depression, the lumbar puncture headache, and the increased peristalsis—are all physiologically explained.

The recognition of complications and the measures employed for their prevention are well described, and the causes of failure carefully analysed. Though Dr. Evans is obviously an enthusiast and views the subject through rose-tinted spectacles, yet his book is a most valuable contribution to a subject of great and increasing importance at the present time. The work is well illustrated.

The Treatment of Fractures and Dislocations in General Practice. By C. Max Page, D.S.O., M.S. (Lond.), F.R.C.S., Surgeon to St. Thomas's Hospital, etc.; and W. Rowley Bristow, M.B., B.S. (Lond.), F.R.C.S., Surgeon to the Orthopædic Department, St. Thomas's Hospital, etc. Third edition. Demy 8vo. Pp. 284 + xiii, illustrated. 1929. London: Oxford University Press. 14s. net.

This edition differs from the second only in being slightly larger. Its chief improvement is the introduction of very clear line drawings alongside the X-ray reproductions which are not very clear. We cannot help thinking that the substitution of similar line drawings for all the X-ray photographs would be an improvement. The main feature of the book is that it follows the practice of the authors, and, as such, illustrates clearly the uses and limitation of plaster-of-Paris in the treatment of fractures. When dealing with this form of treatment, the writing is direct and the note sure, but the descriptions of, and indications for, most kinds of splint treatment are not so good.

The chapter on the operative treatment of fractures is most disappointing. While in a book written for the use of general practitioners no details of the various methods in use should be included, we cannot help thinking that the authors have not realized the importance of impressing upon the general practitioner the principle that early operative treatment is absolutely essential for certain fractures. This particularly applies to intra-articular fractures, and fractures of such bones as the carpal scaphoid. The latter is not mentioned in the chapter devoted to operative treatment, and where it is dealt with, the prognosis and advice as to operative treatment are very undecided. We cannot agree that open reduction without internal fixation has a somewhat limited application. When writing that the chief indication for its use is a fracture-separation of the lower epiphysis of the humerus, it is to be concluded that a supracondylar fracture is meant, as there is no detailed account of epiphysial separations.

It is unfortunate that the only two radiographs showing the application of Lane's plates demonstrate appalling results. We cannot help thinking that a more clear-cut description, founded upon personal experience, of the indications for and use of open reduction would have been of great use to many practitioners. We

cannot agree with the statement on page 110, that fractures about the elbow should be treated in a position of 40 degrees flexion. No warning is given in connection with the treatment of these fractures of the danger of immediate reduction and fixation in flexion. The authors have not yet realized the educative value of reduction of most fractures under an anæsthetic on the X-ray table. Six months' experience of the treatment of elbow fractures under these conditions would convince them that once reduction has been obtained, fixation in full flexion is usually unnecessary.

We cannot see 'eye to eye' with the authors in preferring to pass a pin for axial traction over, rather than through, the os calcis; and similarly, we cannot help thinking that their unfortunate experience of transfixion of the condyles of the femur by a pin for axial traction has been unusual. These are, of course, only minor differences of experience.

The treatment of fractures of the spine with cord injuries is so largely the prevention of renal infection, that something more than a few lines would be helpful in contrasting the different treatments for retention. This section of the book suffers from being much condensed.

On the whole, the book will serve its purpose. It is for the most part candid and practical, so that in many respects it satisfies the needs of a practitioner faced with a difficult fracture. As a rule space is given to consideration of common injuries, both in letterpress and figures. We are confident that this edition will have as brief, and as successful, a life as the last.

Le Diagnostic dans les Affections de la Colonne vertébrale (chez l'adulte). By Professors P. OUDARD, A. HESNARD, and H. COUREAUD (Toulon), with a Preface by Professor SICARD. Medium 8vo. Pp. 256, with 75 illustrations. 1928. Paris: Masson et Cie. Fr. 36.

THIS small monograph represents chiefly the application of modern methods of diagnosis to the problems of disease or deformity of the spine. Radiology and the information derived from the injection of lipiodol now represent the last word in accurate diagnosis of the nature, position, and extent of lesions of the vertebral column. The instinct of the clinician and his interpretation of physical signs and symptoms are now to be supplemented by the precise information derived from modern scientific methods.

The main part of the work, descriptive of normal anatomy and the morbid changes associated with tuberculous disease of the spine, is of great value, because of the simple and clear descriptions and the excellent line drawings and diagrams. The reproduction of actual X-ray pictures is not as clear as might be wished. For this reason the description of deformities of congenital or postural character is not so valuable as might be desired. This is unfortunate because it is in just such conditions as sacralization of the 5th lumbar vertebra, and its distinction from osteo-arthritis, that one would naturally turn to such a monograph as this for help.

Some Principles of Minor Surgery. By ZACHARY COPE, M.S., M.D. (Lond.), F.R.C.S., Surgeon to St. Mary's Hospital, Paddington, and to the Bolingbroke Hospital. Post 8vo. Pp. 159 + xi, with 82 illustrations. 1929. London: Oxford University Press. 10s. 6d. net.

THIS book is a collection of eight papers dealing with surgical principles or certain subjects of minor surgery. The use and abuse of antiseptics is the first of these essays and in it is given a good critical account of the modern application of Listerian principles. The chief value of antiseptics in wound treatment is in dealing with grossly infected superficial wounds soon after their infliction. Acriflavine and Dakin's solution are esteemed to be of the most value. In discussing the treatment of acute inflammation and describing common mistakes in the diagnosis and treatment of acute abscess there is not much that calls for comment. The fourth chapter

is concerned with infections of the fingers, and due importance is given to Kanavel's work. By means of superimposed transparent films useful diagrams are given showing the relation of the palmar connective-tissue spaces to the better defined structures of the hand. Chapters 5 and 6 describe sprains and the ambulatory treatment of fractures. So many different injuries are described, and these so briefly, that it may be questioned whether these chapters are of great practical value. For instance, the so-called sprain of the sacro-iliac joint is dealt with in less than one page, and sacrolumbar sprains in an even shorter space. In injuries of the shoulder and humerus the value of an abduction splint is not mentioned, although this is surely the most important appliance in many such conditions. In speaking of supramalleolar and malleolar fractures, it is perhaps unfortunate that, although in the text it is mentioned that the foot should be placed at right angles to the leg, yet in all the pictures of the plaster-of-Paris splint and its application, the foot is shown in a dropped position.

The next chapter describes the treatment of retention of urine, due to an enlarged prostate, by means of catheters or suprapubic puncture of the bladder, and very great stress is laid on the importance of emptying the distended bladder gradually. The concluding chapter deals with a variety of minor operations—for example, varicose veins, ingrowing toe-nail, and the removal of foreign bodies. We think the book would make a greater appeal if it dealt more with principles and less with details.

A Graphic Guide to Elementary Surgery. By Professor TH. NAEGELI, Bonn. Translated by J. SNOWMAN, M.D., M.R.C.P., with an Introduction by Dr. C. GARRÉ, Bonn. Royal 8vo. Pp. 206, with 322 illustrations (mostly coloured). 1929. London: John Bale, Sons & Danielsson Ltd. 12s. 6d. net.

THIS elementary picture-book of surgery is founded on the assumption that "a picture is worth more than many a thousand words", and has an introduction by the late Professor Garré explaining its purpose of impressing facts on the mind and memory by pictorial representation. We confess that the ideals aimed at have made a strong appeal to us and that we have derived much interest, and may we say amusement, from reading it. It deals only with general principles: anæsthesia, infections, wounds, injuries of bone, tumours, affections of arteries and veins, metabolic disorders, operations, grafting, and methods of examination. The main feature of the book is the composite diagrams—for example, *Fig.* 130 tells the story of pyæmia by means of a temperature chart, a diagram of the infected wound and the spread of infection, with others of the lungs, kidneys, liver, and hollow bones.

Certainly such a simple and graphic method of teaching affords a most valuable introductory book for the student or for the nurse. The method might with advantage be extended for use in a larger text-book.

Kleine Chirurgie. By Professor HANS KURTZAHN, Königsberg. Medium 8vo. Pp. 475 + viii, with 172 illustrations and 1 coloured plate. 1929. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, M. 20; bound, M. 22.

THIS exposition of minor surgery is carried out on the usual lines of such books. Probably no two authors would agree as to the selection of subjects to be included in, or excluded from, the scope of such a work. In the present case the chief principle consists in excluding abdominal surgery or any description of major operations. Curiously enough, the one coloured plate is devoted to the illustration of a very rare condition, viz., erysipeloid affecting one finger. The description of ordinary matters of surgical technique is simple and clear. Injuries and infections of the fingers receive considerable attention, but there is no clear account of the connective-tissue spaces of the hand. The last chapter, which forms an unusual feature of current text-books, deals with prognosis after accidents, with a special view of giving expert opinion in cases arising under the Workmen's Compensation Act.

Hæmorrhoids: their Etiology, Prophylaxis, and Treatment by means of Injections. By ARTHUR S. MORLEY, F.R.C.S., Late Temporary Assistant Surgeon, St. Mark's Hospital for Cancer, Fistula, and other Diseases of the Rectum. Fourth impression, revised and enlarged. Demy 8vo. Pp. 122 + viii, illustrated. 1929. London: Humphrey Milford. 6s. net.

WITH the exception of an additional chapter at the end of the book entitled "An Improved Technique" in which the author adopts the method practised by the late Dr. J. D. Albright, of Philadelphia, and by Dr. C. Elton Blanchard, of Youngstown, Ohio, this book is practically a reprint of the former edition. We have, therefore, no further comment to make upon it except to express our regret that the author has not seen fit to delete the sentence against which we then took umbrage.

Injection Treatment of Internal Hæmorrhoids. By MARION C. PRUITT, M.D., Associate in Surgery, Medical Department, Emory University, Georgia Baptist Hospital and Grady Hospital, etc. Crown 8vo. Pp. 137, illustrated. 1929. St. Louis, U.S.A.: The C. V. Mosby Company. 12s. 6d. net.

THIS small volume, we are told by the author in his preface, represents an attempt to set down his experience with, and to show the value of, the injection treatment of internal hæmorrhoids. After defining the term hæmorrhoids in the opening chapter, the author endeavours in the succeeding one to furnish the reader with a working knowledge of the surgical anatomy of the anus, anal canal, and the lower part of the rectum. Our comment upon this chapter is that these anatomical details will be found to have been more accurately described in any text-book of anatomy. Chapter 3 deals with etiology and prophylaxis; the influence of occupation, age, sex, and heredity is also cursorily discussed. Chapter 4 represents the author's interpretation of the pathological changes which Quénu states take place according to his investigations. Chapter 5 contains a schematic arrangement in the form of a chart which is suggested as a substantial basis for the classification of hæmorrhoids. According to the author's preface, one of his reasons for writing this book was to remedy the confusion that he considers to exist in the minds of the medical profession with regard to the injection treatment of hæmorrhoids, but we are afraid that the classification which he now suggests will make confusion even worse confounded. Chapters 6 to 9 are devoted to the classification of both external and internal hæmorrhoids, their symptomatology, and their diagnosis. Chapter 10 is concerned with sphincteroscopes. Chapter 11 is replete with the opinions expressed by various authors in published papers upon the method of treating hæmorrhoids by injections. Chapter 12 describes the different solutions used by various exponents of the injection method, together with the author's technique for carrying out the treatment. The remainder of the book contains chapters upon care, after-treatment, pathological changes produced by injections, advantages and disadvantages of the treatment, contra-indications, complications, recurrences, and illustrative cases.

We can find no word of praise for this book. It is full of inaccuracies, the names even of well-known authors not escaping mutilation, as, for example, when Sir Charles Gordon-Watson's statistics are attributed to Sir Gordon Eatson.

The Injection Treatment of Varicose Veins. By A. H. DOUTHWAITE, M.D., M.R.C.P., Assistant Physician, Guy's Hospital. Fourth edition. Crown 8vo. Pp. 58 + x. 1929. London: H. K. Lewis & Co. Ltd. 4s. net.

THE author, after an experience of five thousand injections, is more than ever convinced of the great value of this method in the cure of varicose veins. Having tried various alternative solutions, he considers that the quinine and urethane solution is the best. It gives uniformly good results and produces a venitis with more firmly adherent clot than any other drug. Injection is not recommended for the treatment of varicocele.

Imperative Traumatic Surgery, with Special Reference to After-care and Prognosis. By C. R. G. FORRESTER, M.D., F.A.C.S., Consultant, Teaching Staff, Illinois Post Graduate School, Laboratory of Surgical Technique, Chicago, etc. Medium 8vo. Pp. 464 + xxix, with 598 illustrations. 1929. London: William Heinemann (Medical Books) Ltd. 42s. net.

THERE is nothing remarkable in this book beyond the title. Whilst it deals chiefly with fractures, dislocations, and injuries of peripheral nerves, it also includes housemaid's knee and acute osteomyelitis. In the case of fractures of the skull, spine, chest, and pelvis, the possible concomitant injuries to brain, spinal cord, and viscera are only briefly mentioned.

When we are told that after removal of a semilunar cartilage from the knee the patient should be able to walk without the use of an artificial support in from five to eight weeks, and that for traumatic synovitis the treatment should be immobilization of the limb in a plaster cast for six weeks, we can but regret that the hustle characteristic of American life has not been universally adopted by 'traumatic surgeons'.

Lister Redivivus. An Essay on the Undue Prevalence of Sepsis. By A. C. F. HALFORD, M.D., Honorary Consulting Surgeon, Lady Lamington Hospital, Brisbane. Royal 8vo. Pp. 110. 1928. Brisbane: Sapsford Bros. 5s. net.

THE history of the methods of treating wounds and of the antiseptic principles of surgery is a fascinating one, the epic of which still remains to be written. The beginning of the story is vague and nebulous and goes back to times earlier than our earliest records: it continues through the centuries, and even to-day universal agreement on this fundamental subject has not been reached.

More than 600 years ago, before the Battle of Crécy was fought, a famous French surgeon, Henri de Mondeville, affirmed his belief, which amounted in those days almost to heresy, that suppuration was not an essential process in the healing of a wound, and that wounds could, and did, heal by first intention, if treated by the methods he advocated and gave to the world in his book on surgery. The main principles of Mondeville's treatment, the exact reasons for which he did not appreciate, were to remove all foreign bodies from the wound, to avoid probing, to wash it with wine, to avoid exposure to the air by means of early suture, and then to cover it with compresses soaked in wine. The introduction of this novel treatment, which compares very favourably with modern aseptic principles, seems to have caused an immense commotion in medical circles. Mondeville says himself that he had much to put up with, vehement discourses, violent words, perils, and menaces so threatening that he would have abandoned it if he had not had the support of his royal patron, the Count of Valois.

It would appear that Mondeville's method was not his own, but taken from his master, Theodoric, and then elaborated and improved. But where did Theodoric get it? The answer is not given, but the story of the Good Samaritan suggests that something like it had existed for many years. After the death of Mondeville the method was abandoned, and the great Guy de Chauliac, writing fifty years later, speaks of it with disdain. So 600 years elapsed before the illuminating discoveries of Pasteur, and their application to surgery by Lister, gave to the world the true knowledge of the cause of suppuration, and made surgery safe. These epoch-making discoveries ushered in the era of antiseptic surgery, soon to be followed by that of aseptic surgery. This great revolution, though, did not come peacefully. The doctrines of Lister were not adopted quickly and universally. Antisepsis and asepsis: the controversies on these raged to and fro, until the clouds of confusion gradually cleared, and the true meaning of the difference, or want of difference, between the methods has become evident and the medical profession has settled down with complacency to what has become an international standard method.

Now Mr. Halford, in his book *Lister Redivivus*, wonders what Lister would think if he were to come to life again and see how different the methods at present in

vogue are from the ritual he elaborated. He thinks Lister would be sad, miserable, shocked, for in his opinion the world has adopted an unsound, fallible method instead of a sound one. His thesis is that there is too much and avoidable sepsis in surgery and midwifery, all of which could and should be avoided by adhesion to Lister's own methods. From many of Mr. Halford's opinions we may dissent; we may be irritated by his methods of argument, for he hits out hard and often, but at the same time we thank him for calling attention to a most important subject, and for the obvious sincerity and honesty of purpose which shine out from every line. The author is a man of mature age who has been for many years a general practitioner and Medical Officer of Health in Australia, and he speaks out of the fullness of his experience. His book also is introduced by Mr. Hamilton Russell, whose opinion is universally respected, who says of him in a Foreword that "it is the cry of a thoughtful and enthusiastic practitioner of medicine, prompted, I may even say goaded, to a divine discontent at the present-day usages that purport to be the modern and improved expositions of the principles of Lister."

It is not an easy book to read or review because of a lack of clear exposition, which engenders confusion of thought in the mind of the reader and, may one guess, in the mind of the writer as well. The subject raised, however, is so important, and such serious criticism is levelled at the methods at present in vogue, that the book has been read with the care it deserves and, as far as possible, without prejudice.

If we have understood the author aright, the following are the points he makes: (1) There is undue sepsis in medical practice to-day: operation wounds 'go wrong': contaminated or infected wounds, which might have been cleansed by Listerism, are allowed to suppurate: tuberculous abscesses become secondarily infected and parturient women become infected, many dying, and still more undergoing long and painful illnesses. (2) All these could be prevented by using Lister's methods. (3) The modern usage of dressings and instruments sterilized by heat, and without the use of 'antiseptic' chemicals, does not prevent contamination of the wound, either at the time of operation, or in the post-operative period of dressing and convalescence, or in the puerperal or post-puerperal period.

These seem to be the main arguments employed when dissected out of the mass of observations and objurgations contained in *Lister Redivivus*. If these points are examined it seems clear that most, if not all, will at once agree that there is still a regrettable amount of sepsis. What surgeon can deny that sometimes an operation 'goes wrong'? What obstetrician can say that he never sees a case of puerperal sepsis in his practice? Conceding these points, the main argument has to be examined. Would there be less sepsis if all wounds, and all midwifery cases, were treated by Lister's methods, as Lister used them? Mr. Halford thinks there would. There seems to be a very large body of opinion of a contrary character. Is it not clear that a clean wound is to be kept free from contamination with micro-organisms? Is it not better to take such steps as will ensure that none get in rather than to kill them once they have got in? From the point of view of clean operation wounds, surgeons have arrived at the opinion, on good and sufficient grounds, that sterilization of instruments and dressings by heat, and protection from infection of hands and mouth by gloves and masks, give good results.

The treatment of accidental, already contaminated, or infected wounds may be considered more open to argument. Mr. Halford points to the terrible amount of suppuration seen in the Great War, and infers that all the dreadful sights revealed then to military surgeons might have been banished by Listerism. He says: "How powerless aseptic surgery was under these new conditions we can read in the medical records of the Army Medical Services. What is most remarkable about these reports is the ultimate conclusions of the most energetic research workers, that the most that could be done was to subject all infected wounds to continuous irrigation with an antiseptic for long periods." It is true that we were a long time controlling sepsis in war wounds, but this was largely due to the conditions of warfare which compelled wounded to be brought back to the base, and thus delayed treatment for days. In the later stages of the war, things were very different. One of the most

important and fundamental pieces of research was done and published by the late Forbes Fraser: "Primary and Delayed Primary Suture of Gunshot Wounds: A Report of Research Work at a C.C.S." (*Brit. Jour. Surg.*, 1918, vi, 92). This work seems to answer in most respects these criticisms, and though these remarks may perhaps not convince Mr. Halford, they may serve to show him that there is at least another side to the shield. He may be pleased to think that his criticisms may, as they certainly should, cause every surgeon to pause and ask himself if he is doing all he ought. If this results, then the book is justified, and Mr. Halford may be to surgery, as the pearl to the oyster, a precious irritant.

Indigestion: Its Differential Diagnosis and Treatment. By HERBERT J. PATERSON, C.B.E., M.C., M.D., M.A. (Cantab.), F.R.C.S., Senior Surgeon, London Temperance Hospital. Pott 4to. Pp. 153 + viii. 1929. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

IN this book the author has endeavoured to present a practical guide to the differential diagnosis and treatment of indigestion. As the book is intended for clinical use and not for historical research, references to statistics, etiology, and pathology have been omitted, except in so far as is necessary for an adequate understanding of the subject. He first of all attempts a classification of disorders of digestion; this is followed by a chapter on clinical investigation in which he rightly emphasizes the outstanding importance of the history. Under the heading of physical examination a good deal of space is devoted to test-meals and their significance, the author believing that in their proper place such investigations are a great help in the diagnosis of the causes of indigestion. We think that he over-stresses the value of test-meals; gastric analysis cannot be other than an inexact bio-chemical procedure, for not only are the normal variations very elastic, but these variations themselves depend on factors which are constantly changing, and of these the psychic factor may be of great importance, depending as it may upon the reaction of the patient to the extreme unpleasantness of the passage of the stomach tube. In the chapters dealing with gastric and duodenal ulcers and cancer of the stomach we find a repetition for the most part of the descriptions to which we have become so accustomed in the literature, and the same may be said of the chapter on complications.

Under the heading of surgical treatment of duodenal ulcer the author asserts that gastrojejunostomy is a physiological operation; we presume he means by this that it is a chemical, as opposed to a mechanical procedure; for an operation which removes the emptying point of the stomach out of the line of the peristaltic drive of the muscle can hardly be termed physiological. We think the value of his assertion would have been enhanced if he had produced arguments to show that the mechanical factor of a new opening has no significance. The book terminates with a short chapter on the general treatment of disorders of digestion, and an appendix in which various tests, qualitative and quantitative, are described.

On the whole the book fulfils its purpose as a practical guide to the differential diagnosis and treatment of indigestion, but we do not think the author is serving any very useful purpose in presenting it. The matter it contains has already been published in book form on very similar lines; there is nothing new in it, and nothing to stimulate thought along fresh channels. The author is so well known as an authority on gastro-intestinal disorders that we confess to a feeling of disappointment that he should have presented a book which does not carry us any farther than we were ten or fifteen years ago.

Chirurgie de l'Articulation temporo-maxillaire. By L. DUFOURMENTEL, Professor of Maxillo-facial Surgery in the Dental School of France. Medium 8vo. Pp. 228 + x, with 69 illustrations. 1929. Paris: Masson et Cie. 6s. 6d. net.

THIS monograph deals with a subject which enters the province both of the general and of the dental surgeon. The temporo-maxillary joint is difficult to examine either

by palpation or by radiographs, and the latter can only be interpreted with difficulty. The diagrams of the anatomy of the joint are good and clear, although the subdivision of the ligaments is depicted as being much more distinct than is really the case. Arthritis, acute or chronic, and trismus are described fully and on usual lines. The most interesting and valuable chapters are those relating to deformities of the condyle, prognathism, and ankylosis. The treatment of prognathism by resection of the condyle on each side is very interesting, but is open to two critical remarks. What is the late result of excision of both condyles? One would like to know the condition, as regards movements and mastication, five years after the operation. The other point is that, judging from the accompanying illustrations, the amount of prognathism was hardly enough of a deformity to justify the operation in some of the cases.

The chapter to which the general surgeon will turn most frequently is that on ankylosis of the joint. The author claims to have operated upon more then fifty cases with only three recurrences. The line of the articulation is cut by means of a number of adjacent drill-holes and the surfaces are smoothed off. No attempt is made to interpose either muscle or foreign material, and the wound is closed without drainage. Great reliance is placed upon the use of an apparatus (Darcissac) by which almost constant immobilization of the jaw is achieved. This consists of two levers fixed to the two jaws and so activated by an elastic spring as to keep the jaws open. The patient will close the jaw in the act of speaking or swallowing, so that the articulation is kept constantly moved.

Branchial Cysts, and other Essays on Surgical Subjects in the Facio-cervical Region. By HAMILTON BAILEY, F.R.C.S., Surgeon to the Dudley Road Hospital, Birmingham, etc. Crown 8vo. Pp. 86 + viii, with 50 illustrations. 1929. London: H. K. Lewis & Co. Ltd. 5s. net.

THIS short collection of essays on subjects relating to the surgery of the neck is of definite practical value. The diagrams of the possible situation and relations of branchial cysts, which have already appeared in this JOURNAL, are clear and simple.

It is rightly maintained that a correct diagnosis can be made in most cases before operation. Stress is laid on the characteristic appearance under the microscope of the fluid withdrawn by puncture. It always contains cholesterin crystals and epithelial débris. A good description is given of the complete operation for the removal of the thyroglossal tract, after division of the hyoid bone. The complications and diagnosis of a submaxillary calculus form the theme of another interesting chapter. The last chapter is on the preventive treatment of cavernous sinus thrombosis in cases of carbuncle of the upper lip. This, consisting in the ligature of the angular vein, was done in four cases, three of which made a good recovery. The subject is one very inadequately dealt with in current surgical text-books, and the present volume is decidedly opportune.

Le Tractus thyréoglosse. By G. RÉMY NÉRIS, Ancien Interne lauréat des Hôpitaux de Paris (Prix d'Otologie). Medium 8vo. Pp. 170, with 23 illustrations in the text. 1929. Paris: Gaston Doin et Cie. Fr. 30.

THIS prize essay brings together in a very convenient and readable form the salient facts about the embryology, pathology, and treatment of cysts, fistulæ, and tumours of the thyroglossal tract. The very intimate relationship of the budding thyroid duct to the hyoid bone in early development is well shown by a number of sections of embryos. This point is of cardinal importance in understanding the persistence of fistulæ and the recurrence after operations upon cysts. It forms the basis of the Sistrunk operation, which is figured and described, the hyoid bone being divided in

order that the whole tract up to the foramen cæcum may be removed. A small quiescent cyst not causing symptoms should be left alone. The drawings and microscopical sections are very clear and convincing.

Gelenkerkrankungen. Einführung in die Pathologie und Therapie. By Dr. ERNST FREUND, Vienna. Royal 8vo. Pp. 497 + xii, with 88 illustrations in the text. 1929. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, M. 32; bound, M. 35.60.

THIS book, which aims at giving an introduction to the pathology and treatment of joint disease, is a general survey of the subject written by a physician. He is fully aware of the size of his subject and its many complexities, and he strives to bring together the many workers and the results of their labours in this compact and practical book. It makes no pretensions to be a surgical text-book. In fact there are only two pages devoted to surgical treatment. But the author makes a strong and an unanswerable appeal for closer co-operation between the physician and the surgeon, in order that knowledge of the scope and results of surgical intervention may be better appreciated.

The book is remarkable rather for the large number of subjects with which it deals than for the completeness with which any one subject is discussed. Every possible disease in which the joints are affected is described. The influence of metabolic disorders, nervous diseases, and diseases of the endocrine glands is discussed at some length. There is a summary of all the methods of treatment, with a short indication of the scope of each. The illustrations, which are not very numerous, are good and clear, but their value would be greatly increased by the addition of an appropriate legend to each, instead of compelling the reader to search for the reference in the text.

Die Vor- und Nachbehandlung bei Chirurgischen Eingriffen. By Dr. M. BEHREND. Second edition. Post 8vo. Pp. 115, with 5 illustrations. 1929. Berlin: Julius Springer. M. 4.80.

THE appearance of a second edition of this small and practical book so soon after the first is sufficient evidence that it has been found of value. It is remarkable how much is contained in so small a compass. For this reason and also because of its completeness and simplicity it will probably continue to prove of value to students, nurses, and house surgeons, in reminding them of special precautions to be taken before operations, and special treatment necessary afterwards.

Œsophageal Obstruction: its Pathology, Diagnosis, and Treatment. By A. LAWRENCE ABEL, M.S. (Lond.), F.R.C.S., Assistant Surgeon to the Cancer Hospital, London. Super Royal 8vo. Pp. 234 + xi, illustrated. 1929. London: Humphrey Milford. 30s. net.

THIS book incorporates the Jacksonian Prize Essay of 1925 revised, enlarged, and brought up to date, and a Hunterian Lecture delivered in 1926. It is well and profusely illustrated, and is written in an easy and entertaining style. It appears to contain an account of all that is known about the œsophagus—its anatomy and deformities; the methods by which it should be examined; its functions, both natural and perverted; and a detailed account of its diseases. Its tone is essentially practical, and the author gives a very lucid and concise idea of his own opinions, as well as mentioning the various alternative theories and methods. At the end of every chapter is a most extensive bibliography, and this feature in itself renders the book very valuable for reference. While it cannot be claimed that there is anything strikingly original in the book, yet it must be appreciated as a most exhaustive account of the present state of knowledge of the œsophagus. In regard to the great problem—the treatment of carcinoma—the author has nothing very encouraging to offer. Radium is mentioned as a possible palliative agent, but all his faith is pinned on excision of the growth. It is suggested that the difficulties presented by operations upon the stomach and colon are strictly comparable with those in the case of

the œsophagus, and that, just as these difficulties have been overcome in the former, so, given "more adequate education and earlier investigation of cases", they may be overcome in the latter. We must be pardoned if we assert that Mr. Abel's statement that under these conditions malignant disease of the œsophagus "will be the most favourable type of cancer with which the surgeon has to deal" appears more like a pious hope than a justifiable conclusion.

Le Cancer primitif du Poumon: Étude anatomo-clinique. By RENÉ HUGUENIN, Ancien Interne lauréat des Hôpitaux de Paris. Preface by Dr. G. ROUSSY. Royal 8vo. Pp. 330 + iv, illustrated. 1928. Paris: Masson et Cie. Fr. 50.

THIS is a monograph dealing with primary carcinoma of the lung from an anatomical, pathological, and clinical point of view. The author describes the sites and appearances, macroscopic and microscopic, of the different forms of carcinoma. He describes the course of the disease, the clinical features, the methods of diagnosis, and finally the forms of treatment and the results of radiotherapy. A useful bibliography is attached.

Les Abcès du Poumon. By MICHEL LÉON-KINDBERG, Médecin des Hôpitaux de Paris. Crown 8vo. Pp. 134, illustrated. 1928. Paris: Masson et Cie. 1928. Fr. 14.

THIS is one of the manuals of practical medicine and surgery which are published from time to time by the house of Masson. The author discusses in a useful and concise manner the etiology, diagnosis, pathology, and treatment of pulmonary abscess. It is a very useful résumé of the subject, and has a good bibliography.

Le Cancer: Maladie des Cicatrices. By AUGUSTE LUMIÈRE, Correspondant de l'Institut; Correspondant de l'Académie de Médecine. Preface by Professor L. BÉRARD, Membre Associé de l'Académie de Médecine, etc. 8vo. Pp. 287 + ix. 1929. Paris: Masson et Cie. Fr. 18.

THE author gives a comprehensive analysis of the literature on the etiology and treatment of carcinomata, and bases his own theories on the conclusions which he draws. Tumours are divided into four groups: (1) Inflammatory hyperplasias; (2) Tumours developed in persistent embryological cell nests; (3) Tumours of connective tissue; (4) Malignant epithelial tumours. It is the fourth group which forms the subject matter of this book. The author believes that four conditions must be present before a malignant epithelial tumour can develop: (1) Delay in the healing of an injury, and the formation of a cicatrix; (2) A latent period of about twenty years; (3) Secondary trauma to the cicatrix to start off the malignant process; (4) A suitable humoral medium. This theory is summed up in the phrase of 'no cicatrix, no cancer'. Doubt is cast upon the value of statistics based on death certificates, and also upon any theory invoking a bacteriological cause of cancer.

The Medical Museum: Modern Developments, Organization, and Technical Methods based on a New System of Visual Teaching. By S. H. DAUKES, O.B.E., M.D., D.P.H., Director of the Wellcome Museum of Medical Science, affiliated to the Bureau of Scientific Research. An amplification of a Thesis read for the degree of M.D., Cambridge. 10 in. × 7 in. Pp. 172, illustrated. London: Wellcome Foundation Ltd. Printed for private circulation.

THE Wellcome Historical Medical Museum in Wigmore Street, London, W., is known to most medical men; it is a unique treasure house of specimens and documents which illustrate the history of medicine. The Wellcome Museum of Medical Science is almost unknown, and yet for medical men it is far more important than the

other, for it seeks to give a graphic representation of the present state of modern medicine. This great addition to the medical institutions of London has grown up so quietly just off the bustle of Euston Road that its existence has almost escaped notice. The book which its curator, Dr. S. H. Daukes, has written about it is thus most opportune.

There is no doubt about it that in the opinion of most practitioners the museums attached to medical schools have outlived their period of usefulness and might well be scrapped. This opinion is also shared by many teachers, particularly those who have to do with the experimental branches of medicine. There is something wrong with medical museums, and the question which many are asking is: How can they be made alive? Dr. Daukes has not only asked this question; he has answered it by bringing into being—through the unstinted generosity of Dr. Henry Wellcome—a great medical museum of a new kind and based on novel lines. Dr. Daukes holds the opinion that pathological specimens can serve a useful purpose in medical education only when they are given their proper setting. They must not be separated from the picture of the patient and his disease. Take a case of endocarditis; the heart with the aortic valves studded with vegetations is shown, but with it is given the photograph of the sick boy from whose body the specimen came. The temperature chart and pulse-rate are shown beside the patient. A graphic picture of the heart symptoms is set beside the chart. With the case is placed the boy's tibia—the site of an acute osteomyelitis. Cultures of organisms grown from the tibia and blood form part of the exhibit. In brief, Dr. Daukes's aim is to make the medical museum the central department of a medical school where all branches of knowledge are made to focus their combined efforts to unravel the nature of a disease, exemplify its symptoms, give the means of diagnosis, set forth graphically the modes of treatment, and, where possible, illustrate the methods of prevention. Some diseases lend themselves to this graphic method of illustration better than others. Tuberculosis and syphilis may be taken as examples. Both these diseases are fully illustrated in the new museum by means of photographs, drawings, models, and actual specimens; the modes of infection, the infective organisms, their isolation and identification; the symptoms, diagnosis, treatment, and prevention are all portrayed. The visitor, be he layman, medical student, or medical practitioner, has set before him a graphic representation of all that is known concerning these particular diseases. The whole field of human disease is brought within the scope of this new museum.

The merits of such a system are obvious; but we must not forget that its attainment is difficult and costly. Not everyone has the resource and happy ingenuity of Dr. Daukes in designing the means which will bring before the student the full story of a disease told graphically; only institutions which can command artists, modellers, and technicians are in a position to undertake such a scheme. Medicine is progressive; every day sees an addition to knowledge, and the exhibits have to alter as knowledge grows. There has to be a continuous struggle to keep up to date; there have to be repeated periods of scrapping; there must be almost unlimited space for exhibition, and apparatus of the most costly kind. There must be a most liberal supply of expensive cases and of exhibition stands. Labels have to be changed or improved. Literature has to be surveyed and abstracted month by month; a great and efficient staff has to be maintained. Granted all these conditions, with an inventive and resourceful brain behind all, such a desirable national institution as the Wellcome Museum of Medical Science becomes possible. Such a museum is a great engine of medical education, and represents medical propaganda of the best kind. Clearly, to carry out such a scheme in its complete form is beyond the financial resources of most medical schools; but without a doubt it is an ideal which every school should aim at.

Dr. Daukes is also alive to another function of museums beyond that of educating medical students. They are also institutions which have to do—or should have to do—with the increase of knowledge. They should cater not only for the needs of students proceeding to examination, and the needs of men in practice, but also for the needs of men who are engaged on research. For research the graphic illustration of current knowledge is useless; only original documents—the actual specimens—

are of value. The research museum and the teaching museum have to serve different purposes and must be organized on separate lines. That is no reason why our larger museums should not aim at serving both purposes, but it would be a misfortune if the possibilities of museums as powerful instruments of research were lost sight of.

Dr. Daukes's book, excellently printed, gives a history of the Museum he has done so much to create. It does more: it supplies curators of medical museums with much technical information for which they will be grateful.

A Surgical Diagnosis. By J. L. DONHAUSER, A.B., M.D., F.A.C.S., Clinical Professor of Surgery, Albany Medical College (Union University). Royal 8vo. Pp. 799 + xxvii, illustrated. 1929. New York and London: D. Appleton & Co. 42s. net.

THE purpose of this book is to set forth systematically and in great detail the methods—history-taking, clinical examination, and laboratory and radiological data—whereby a diagnosis may be established in the surgical conditions affecting all parts of the body. It is based on nearly twenty years' experience in the teaching of surgical diagnosis, and is intended primarily for students, but also for house officers and general practitioners. It is a very thorough and comprehensive work, the compilation of which has entailed much labour. It is a monumental example of one method of clinical teaching, the 'list' method, which appeals to some, but certainly not to all intellects. The student who sets out to learn clinical surgery by preparing lists of all the possible diseases of every individual organ will revel in this book to his heart's content, for these lists are irreproachable in their completeness. Their appalling length will, however, fill with dismay a mind not so pigeonholed; and they will leave unmoved the student who is pinning his faith to an underlying uniformity in pathology in all organs, modified here and there by the anatomical or physiological peculiarities of the affected part.

The contents are divided up into twenty-four sections, ten of which deal with abdominal surgery, four with conditions affecting the extremities, and the remainder with diseases of the head, neck, thorax, urogenital system, the general consideration of the examination of patients, the infections, and the tumours. In each section a complete list of all the diseases affecting that part is given, and in the following pages a few paragraphs are devoted to each disease in turn, with a brief description of the salient features differentiating it from the others. At the end of most of the sections there is a large table of differential diagnosis. Most of these tables are too cumbersome and complicated to be instructive. One feels that the object of the author has been to leave nothing out, and one fears that his success renders the book too burdensome for the average student. Except for those capable of incredible feats of memory, it is more likely to be of value as a book of reference than as a text-book.

Medical Adventure: Some Experiences of a General Practitioner. By Dr. ERNEST WARD, M.D. (Cantab.), F.R.C.S. Crown 8vo. Pp. 291, with 10 illustrations and a portrait of the Author. 1929. London: John Bale, Sons & Danielsson, Ltd. 8s. 6d. net.

THE present volume deserves a warm welcome from all medical readers. It represents a much more serious contribution to medical literature than is implied by its title. Many of the chapters are articles written for the *London Hospital Gazette* to bring before students the realities of general practice. The great value of the articles consists in the fact that they are the outcome of real experience, and that the problems are viewed from the point of the man in family practice. Chiefly they deal with commonplace diseases—for example, measles or mumps—and there is a preponderating number of chapters on skin diseases, both rare and common. The few chapters on surgical subjects are all full of acute observation and are illuminated by great humour. As an example of this, in the chapter on carcinoma of the rectum we are told of a case of apparently hopeless ulcerating growth of the bowel

which was cured by anti-specific remedies. The patient celebrated his recovery from imminent death by attending no fewer than three 'revival meetings' in one day. We wish that Dr. Ward would give us further articles dealing with the diagnosis, treatment, and results of surgical conditions, as seen by the family doctor.

Catalogue of Lewis's Medical and Scientific Circulating Library. Part I, Authors and Titles. Part II, Classified Index of Subjects, with Names of Authors who have written upon them. Revised to the end of 1927. Demy 8vo. Pp. 576. 1929. London: H. K. Lewis & Co. Ltd. 15s. net. (To Subscribers 7s. 6d. net.)

MESSRS. LEWIS have carried out a work of great value and importance in the foundation of their circulating medical library. By this means many an isolated practitioner has been able to get at otherwise inaccessible books, and also the smaller medical libraries can greatly enlarge the scope of their usefulness by affiliation with this organization. The present catalogue is not only a list of all the books available, but is accompanied by an index showing which books are available on any particular subject, and the author's name and date of publication. This catalogue and index greatly enhance the value of Lewis's library.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXIX (Vol IX, Fourth Series), No. 1, January, 1929. Medium 8vo. Pp. 126, illustrated. 1929. London: Lancet Ltd. 12s. 6d. net.

AMONGST other articles this number contains: "Notes on the Etiology of Appendicitis", by W. H. Bowen, in which the author concludes that the main etiological factor in appendicitis is stagnation in the appendix; "Hydatid Cyst of the Kidney", by R. P. Rowlands; and "Visualization of Bile-ducts after an Opaque Meal", by J. F. Venables.

Report on Fourth International Congress of Military Medicine and Pharmacy, Warsaw, Poland, May–June, 1927. By COMMANDER W. SEAMAN BAINBRIDGE, C-F., United States Naval Reserve; Member of Permanent Committee. Demy 8vo. Pp. 248 + ix, illustrated. 1927. Wisconsin: The Collegiate Press.

THIS report has been compiled, presumably for the American Government, by a member of the Permanent Committee. It is difficult to assess the status of an International Congress to which France sent over 70 delegates, Great Britain 7, and the United States 5.

It is clear that subjects of fundamental interest in military medicine were under discussion, including the evacuation of wounded, head injuries with all their complications and sequelæ, and the therapeutics of the arsenobenzols. Apart from the immense range of these different subjects, it is difficult to assess the value of the conclusions of the Congress, because no one of the British delegates can boast of large clinical experience of the subjects under discussion.

It would seem that the vision of Belgium, who called the first Congress in 1921, has been amply justified, but if the delegates at these Congresses are granted opportunities of self-education in the care of the sick and wounded, it is not easy to understand why the representatives from Great Britain are limited solely to regular members of the three services.

Reflections and Operations. By Sir JOHN O'CONOR, K.B.E., M.A., M.D. (Dublin), late Senior Medical Officer, British Hospital, Buenos Aires. Edited by BEATRICE and MAY O'CONOR, with a Foreword by HERBERT J. PATERSON, C.B.E., M.C., M.D. (Cantab.), F.R.C.S., Senior Surgeon, London Temperance Hospital. Demy 8vo. Pp. 361 + xxxvi, with 4 plates. 1929. London: Baillière, Tindall & Cox. 21s. net.

OVER twenty-five years ago the reviewer, like many others, was attracted by the writings of O'Conor, of Buenos Aires, and now welcomes the collection of his numerous published papers in one volume. O'Conor left England for the Argentine

in 1889 as doctor to a mining company, on the failure of which he was appointed House Surgeon to the British Hospital in Buenos Aires, soon becoming its Senior Medical Officer—a position he retained until his death.

Few young surgeons can have had such unrivalled opportunities of major surgery, and O'Conor soon proved his worthiness to be entrusted therewith. He was a 'general' surgeon in the widest sense of the term, and his writings, covering practically the whole field of surgery, display marked originality, extraordinary breadth of view, clear thinking, concise description, sound common-sense judgement, fearless criticism, and hatred of all shams. His great practical experience justifies his unusually dogmatic style in teaching, and everywhere his writings abound in most useful practical hints and 'tips'. Many of his views run counter to those usually regarded as orthodox—for example, his preference for anterior rather than posterior gastro-enterostomy, his rule "never to quit the abdomen in acute appendicitis without removing the appendix", his strictures upon the use of early massage and movement in the treatment of fractures, and his spirited defence of Whitehead's operation for hæmorrhoids. Moreover, his advocacy of alcohol both as an article of diet and in the treatment of disease would not be approved by our numerous temperance associations. No one can fail to enjoy and to be encouraged by a perusal of this volume, and to be the better for the introspective self-criticism it must spontaneously evoke.